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THE  
JOURNAL  
OF  
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NORMAL AND PATHOLOGICAL.

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## THE LYMPHATICS OF THE WALLS OF THE LARGER BLOOD-VESSELS AND LYMPHATICS. By GEORGE HOGGAN, M.B. Edin., and FRANCES ELIZABETH HOGGAN, M.D. Zurich, M.K.Q.C.P. Ireland. (PLATE I.)

THE structures which we are about to describe will be found to have most important bearings upon the conceptions now entertained of the physiology of the lymphatic system, as well as upon its anatomy; for none of the functions seriously attributed to it at the present day will be found capable of explaining its existence and relations in the walls of the larger lymphatics, and the only function which, in our opinion, may hypothetically be held to account for its presence there, will also apply equally, in general, throughout the body, giving an additional importance to the lymphatic system.

The presence and peculiar position of the lymphatics within the walls of the larger lymphatics themselves appear, with our present conceptions of the physiology of the system, to be nothing more nor less than an anatomical curiosity. Although the same thing cannot be said of the lymphatics of the walls of arteries and veins, and however great a dissimilarity may, at first sight, appear to exist in the relation of the lymphatics to the different structures which enter into the different coats or layers forming the walls of the three kinds of vessels, we shall be able to recognise with tolerable clearness that the plan of distribution is the same in all three. It is somewhat curious that, notwithstanding the fact that in former days the idea of the existence of lymphatics within vascular walls (and indeed everywhere) had its origin in this country as a hypothetical deduction from certain experiments on animals performed by the Hunters and

their successors, that idea having been overthrown, or been supposed to be overthrown, by the experiments performed by Majendie, it has at length disappeared from all the latest English text-books on anatomy and on physiology, as far as we have yet been able to ascertain. Even Carpenter, whose work is supposed to refer to every physiological question under the sun, makes no allusion to this one; and it has been left in these days to Continental writers to keep up the Hunterian legend, and to Continental investigators to demolish the Hunterian hypothesis by applying to it the test of a practical investigation.

Of those who have practically investigated this question, Professor Sappey is certainly the most important, and no more recent authority could be quoted. At page 797 of his *Anatomie Descriptive*, published in 1876, he says:—"The internal membrane of the circulatory apparatus has been considered as a closely serried plexus of lymphatic capillaries. This opinion, emitted by the illustrious Hunter, was published at first by his pupil Cruikshanks, then by Mascagni, and afterwards by a great number of authors. Lauth, by showing such capillaries on the walls of the heart, attracted new partisans to the doctrine. Shortly afterwards, Breschet admitted that the plexus discovered by that anatomist becomes prolonged upon the whole extent of the circulatory apparatus. The employment of the mercurial injection tube, said he, allows us to recognise that the interior of the whole vascular system is formed by lymphatic vessels.

"Proposing to myself to study these vessels, I have explored with the point of the tube the walls of the arteries and veins, fully convinced that I was about to see a plexus of numerous capillaries rapidly display itself at the first juncture. My surprise has been great not to find them anywhere upon the arterial and venous walls belonging to the different parts of the body. I have never been able to see the smallest canaliculi. Other investigators have taken up this research, but with no better result. We may, therefore, admit that the blood-vessels are destitute of lymphatic vessels." To the foregoing we may add, that nowhere in his works can we discover that Sappey makes even the most distant reference to lymphatics within the walls of the larger lymphatic vessels.

Contemporaneously with Sappey's earlier researches into the lymphatic system, we have the writings on this subject of the great French anatomist Cruveilhier, whose opinions, however, seem to be derived more as deductions from hypotheses than as ascertained anatomical facts. The opinions of Cruveilhier on the present question are no exception to his usual practice. At page 120 of the third volume of his *Anatomie Descriptive*, published in 1851, he says:—"We have as yet only partially found the lymphatic network of the internal membrane of the veins and arteries, but the analogy which

exists between the internal membrane of the serous surfaces and the internal membrane of these vessels is such, that I have no doubt whatever of the identity of the results as regards the lymphatic plexus. I have, moreover, met with special lymphatics of the aorta, injected with blood, in several cases of alteration of the wall of this vessel." At page 136 he further states :—"Lymphatic vessels of an excessive tenuity, analogous to the vasa vasorum of arteries and veins, probably arise from the walls of the lymphatics."

With reference to these opinions of Cruveilhier, we may at once state that we have never yet seen the slightest vestige of a lymphatic within the intima of arteries or of veins having a muscular coat; and there is every reason to suppose that the lymphatics injected with blood in the aorta were only blood-vessels, and not lymphatics. Further, there is no more tenuity in the lymphatics which, he says, *probably* exist in the lymphatic wall, than in the lymphatics elsewhere in the body.

The German anatomists seem to give little attention to this question, their remarks being evidently borrowed from the French. Henle, in his *Handbuch der Anatomie*, page 407, says :—"Hunter, Mascagni, and Breschet had looked upon the inner membrane of the blood-vessels as a structure rich in lymphatics, and had even described it as consisting of coils of lymphatic vessels. For the intima of the blood and lymphatic vessels this opinion has long ago been given up; even in connection with the endocardium, their existence has been disputed by Sappey." Eberth, who, with Belajeff, published an account of the lymphatics of the endocardium in Virchow's *Archiv*, repeats the opinion he expressed there, in his article on the blood-vessels, in Stricker's *Handbook*, vol. i. p. 266, as follows :—"Lymphatics have not hitherto been traced into the coats of the blood-vessels. The lymphatics of the endocardium only extend as far as the semi-lunar valves."

In our own country, the latest opinions we have been able to discover, to which any weight can be ascribed, are those of Lane, in the article "Lymphatics," in Todd's *Cyclopædia of Anatomy*. At page 216 he says :—"With respect to the universal network of lymphatics, attributed to the lining membrane of the heart, and to that of the arteries and veins, I cannot admit that the injection of a few minute canals with quicksilver on the lining membrane of the heart of the horse by Lauth, and similar injections by Cruveilhier and Bonamy, can be received as demonstrations." At page 217, with a caution more characteristic of the investigator than of the compiler, he justly states that his own anatomical and physiological investigations lead him to consider that the extent to which the lymphatics are supposed to exist in the organism must be greatly curtailed. He also believes that "anatomists have not yet freed themselves from the influence of the Hunterian views which supposed ubiquity for the lymphatics." An example of the influence which he deprecates, bearing upon the present question, is to be found in a previous volume, under the article "Absorbents," by J. Hart, who gravely infers, from the absorption of blood coagula from within the ends of ligatured arteries, that the presence of absorbents is thereby proved in every part of the arterial

system. This is the Hunterian doctrine pure and simple. The disappearance of blood-clot is called absorption. Only lymphatics, he assumes, absorb; therefore, the absorption of blood from the interior of arteries demonstrates the existence of lymphatics on the inner surface of the whole arterial system.

The foregoing remarks, it will be understood, only refer to the intrinsic lymphatics actually within the walls of the blood and lymph vessels, and they have no bearing upon the question of lymphatic sheaths, or of companion lymphatics of blood-vessels. The former are still much believed in at the present day, more especially in connection with the lymphatic sheaths or spaces which are supposed to surround the blood-vessels in the brain. To all statements in regard of the existence of such structures we give an unqualified contradiction. The facts which were supposed to prove them were artificial productions, and already, in our article on the lymphatics of the pancreas,<sup>1</sup> we have explained the cause of such artificial appearances, and described a method by which they may be produced at will under the microscope, where every step in the process may be watched, and the relation of such imitation lymphatics with the real lymphatics made perfectly clear, both of them being demonstrated within the same preparation.

The question of companion lymphatics of blood-vessels, although hitherto left out of consideration in the short historical sketch we have given, has really intimate connection with the present subject. It is only in the walls of the largest blood-vessels that we expect to meet with intrinsic lymphatics; but with a set of smaller veins and artery we often find companion lymphatics, forming, it may be, a more or less open and irregular plexus round the group, as if placed there to drain off excess of fluid escaping from the blood-vessels. This condition becomes even accentuated where no collecting lymphatics lie in the vicinity of the group—a condition which we have explained at some length in our article on the lymphatics of the urinary bladder.<sup>2</sup> As far as their function of drainage is concerned, there is thus intimate resemblance between the extrinsic plexuses now referred to and the intrinsic lymphatics which

<sup>1</sup> *This Journal*, July 1881.

<sup>2</sup> *Ibid.*, April 1881.

form the special subject of this paper. It is only when these latter are found on the internal surface of veins and lymphatics that the resemblance ceases, and a new function seems indicated that is unrepresented in the extrinsic or companion lymphatics.

Before proceeding further, let us give a short account of the method of preparation, and of the structures we have examined. The present study does not pretend to be an inquiry into the comparative anatomy of the lymphatics of vascular walls in many different classes of animals. We are more concerned with establishing the fact of their existence, on account of the bearing it has on the general function, or one particular function, of the lymphatic system; for it must be some function in addition to those of modified absorption, and of a peripheral drainage system, which calls for the existence of intrinsic collecting lymphatics upon the inner surface of the thoracic duct itself. We start with the knowledge that in the smaller vessels of the larger mammals, as in the largest vessels of the smallest mammals, the structures we describe do not exist. We therefore intentionally choose the largest vessels in the largest mammals at our disposal (principally the goat and horse) as the best materials for our research, as in them we are certain to find the lymphatic system at its fullest development, the various tissues forming layers in the vascular walls being most distinct, and the relationship of the lymphatics to the layer most clearly defined.

In the case of every vessel we wish to examine, the use of our histological rings is absolutely necessary.<sup>1</sup> As, moreover, it is necessary to get rid, as much as possible, of the flocculent tissue forming the external coat, or *tunica adventitia*, of the veins and arteries, it is not advisable to dissect these out from the tissues in which they lie buried, but, if strong enough, to tear them out, as by this process the vessels come away, deprived in most cases of this flocculent coat and of the fat cells which are lodged often within it. This is always possible in the case of the systemic arteries and the pulmonary arteries and veins. Elsewhere the smaller veins and largest lymphatics must be dissected out, and the loose fibre and fat cells subsequently removed carefully from their outer surface after they are stretched upon the rings.

<sup>1</sup> *Journal of the Royal Microscopical Society*, July 1879.

Within this adventitia no lymphatics ramify, but they only pass through it to ramify in the solid gelatinous matrix of the wall lying internal to the adventitia, and on the outer surface of a muscular layer, when such exists. The adventitia prevents the passage across it of the solutions of silver and gold used by us to demonstrate the lymphatics; it is therefore necessary to remove it before the solutions are applied. Whether this adventitia be removed by tearing it out, in the case of strong vessels, or picking it away with forceps, in the case of the weaker walls of lymphatics and veins, after these have been stretched upon the rings, the greatest care must be taken not to touch the cleared surface with the fingers lest it be made greasy, and the solutions be thus prevented from acting fairly upon the tissues beneath. The vessels having been removed and slit up with scissors, portions of the wall are placed on the rings, with the internal or endothelial surface of the vessel turned outwards, for the outer surface of the tambourine thus formed. From this surface the endothelium, or lining cells, and the serum are removed by passing the edge of a sharp scalpel lightly across it; if the opposite surface have still any of the adventitia adhering to it, that ought also to be removed by a pair of sharp-pointed forceps. If the tissue is too thick to be transparent under the microscope, as in the case of the aorta of a horse, it is easy to remove either the inner or outer half of the wall, by splitting it off with the point of a sharp scalpel, leaving upon the rings that surface in which it is intended to demonstrate the lymphatics.

. The foregoing conditions having been obtained, the rest is very easy. A 2 per cent. solution of nitrate of silver is poured rapidly on and off the surface or surfaces we wish to prepare. After a few minutes' exposure to ordinary light, the tissue is well washed with ordinary water, and a 1 per cent. solution of chloride of gold allowed to remain in contact with one or both surfaces which have been silvered. The whole is then well washed, and the requisite exposure effected in a not too bright light, until, by repeated examination under the microscope, we are able to ascertain whether the preparation is likely to prove successful. When it has attained the requisite development, it is clarified in the usual manner by essential oil or glycerine, and excised and mounted as a permanent preparation.

We have already remarked that, although at first sight the position, distribution, and relationships of the lymphatics in the coats of arteries, veins, and lymphatics respectively may appear to be entirely different, they yet follow one general law in their arrangements; and to understand that general law may prove to be of much more importance than merely noting their different appearance in each of the three structures. To understand it, we must trace the development of these three structures, and the gradual formation of the different tissues which compose them, and which mainly regulate the position and character of the lymphatics within their walls. We must get rid of that artificial division and subdivision into many layers, which is so well calculated to puzzle would-be anatomists, and reduce their description to the simplest form. When we trace the arteries and veins to their most minute or developmentally simplest condition as capillaries, and the main efferent lymphatics to their simplest form of channels of origin, we find their walls constituted only by one thin layer of endothelial cells, hollowed out originally, in the case of the former, by vacuolation, and subsequently enlarging their calibre in all three cases by the interposition of new cells between the cells originally forming the wall. With that layer of endothelium, or lining cells, we may state, once for all, that at no period have the lymphatics any connection whatsoever. As the different vessels increase in size and importance, we find their walls strengthened by the addition, externally, of a thin coating of gelatine, elaborated possibly by the cells forming the wall, as an oyster elaborates the shell around its body. This gelatinous coat forms a very important factor in the present question, inasmuch as within it, as within a hyaline matrix, the muscular elements, with the branched cells, the blood-vessels, lymphatics, and nerve bundles, lie in special relations to each other, according to the character, size, and condition of the various vessels in which they are to be found. That relation seems invariable in the walls of the higher developed vessels of either of the three kinds, and may be shortly stated in order as follows, passing from within outwards:—

1. The endothelium of flat cells lining the inner surface of the wall.



2. A certain thickness of clear gelatinous tissue or matrix, in which lie numerous branched cells, probably wandering cells, which by many anatomists would be called the fixed branched cells of the connective tissue. No blood-vessels or nerves appear in this layer.

3. The muscular coat composed of one, or many, layers of muscle cells, with their nerves and blood capillaries, but destitute, as a rule, of lymphatics.

4. An outer coating of gelatinous tissue, which is the special seat of the lymphatics and of the larger blood-vessels and nerve bundles, and containing many branched cells.

5. The loose gelatinous fibres of the tunica adventitia, which, of all the structures of the body, perhaps best merits the term fibrous tissue. It is composed of a felting of long loose fibres, completely disassociated from each other, and woven at every possible angle to each other around the vessel, from which they may easily be torn off as flocculent bundles. Lying within this feltwork many branched cells and fat cells may be found, but no lymphatics. The lymphatics, with the larger blood-vessels and nerves, pass through it to ramify in the solid matrix of the fourth or outer layer of gelatinous tissue, including everywhere under that head both yellow elastic and white fibrous tissue, which are only modifications of the same substance, and undistinguishable by the silver method of preparation.

In the foregoing scheme, as applied to the larger arteries, which possess intrinsic lymphatics within their walls, the position assigned there to the lymphatics is invariable, just as the coating of muscle cells is invariable, even if modified or unequal in thickness on different parts of the periphery of the same vessel.

In fig. 1 we have an example of the manner in which the efferent lymphatics have their origin, so to speak, in a network of collecting lymphatics lying within the outer layer of gelatine upon the outer surface of the muscular coat. In no case whatever, although we have examined many preparations for it, have we met with lymphatics penetrating the muscular coat, and appearing on its inner surface. This is exactly the contrary of all hypothetical suppositions in the past, as well as of the reputed facts which Sappey has so completely demolished; it is even possible that this absence of lymphatics from the internal

portions or layers in the walls of arteries, has some bearing upon the question of atheromatous formation or degeneration in the same locality.

In one respect, this absence of lymphatics from the inner coats of arteries, while they may exist in the outer coats, quite agrees with the general function of peripheral drainage adjuncts, which appears to be the principal use of the lymphatic system throughout the body ; but, when this is granted, it only makes more incomprehensible the fact that, in non-muscular portions of the walls of the larger veins and lymphatics, typical collecting lymphatics are often to be found in great profusion, as shown in figs. 5 and 6.

According to the views held at the present day, it would not be so very strange that collecting lymphatics should appear close to the inner surface of arteries, in immediate proximity to a freshly elaborated or purified blood or blood serum on its way from its centres of purification to supply the peripheral elements with nutrition. On the other hand, why, according to the same views, these collecting lymphatics should appear upon the inner surface of veins and lymphatics, close to an exhausted and impure blood or lymph on its way from the periphery to the purifying centres, impeding its progress towards those centres by draining it away from the canals which form its shortest route thither, is sufficiently puzzling. Yet this latter condition everywhere exists. The best explanation of these apparent incongruities is probably an anatomical one, unconnected with the functional conditions we have referred to. In portions of the walls of veins and lymphatics furnished with a muscular layer, or coat, and approximating therefore anatomically to the invariable muscular coat of arteries, we have hitherto failed to find lymphatics on the inner side of such muscular portions of the walls, as if the contractile action of the muscle cells was detrimental to the development or existence of lymphatics between these cells and the fluids upon which they exercise a pressure. At all events, if this be not an explanation, it is an interesting coincidence which deserves to be taken into consideration.

We were early struck with the fact that, while upon one portion of the wall of a large vein or lymphatic the muscular layer would be largely and regularly developed, as at fig. 4, yet, on the

opposite point in the periphery of the same vessel-wall, and at the same level, the muscular layer might be altogether wanting, where only innumerable branched cells studded the matrix of gelatine forming the wall external to the endothelium. The change from branched cells to muscle cells was a very gradual one, and there could be little doubt that the one was developed from the other, as numerous instances of such cells undergoing the different stages of change, from the one to the other, were to be seen in our preparations. After careful observation of surrounding circumstances, we seemed to have arrived at the explanation of this difference in the wall of the same vessel, or at all events at one of the explanations of this condition. When a large vein or lymphatic is attached to some solid structure, as, for example, the vena azygos or receptaculum chyli upon the vertebral column, that portion of the wall which is attached or fixed to the column in such a way as to be rendered immovable, and incapable of contraction, would generally be found to be destitute of muscle cells, but to contain innumerable branched cells, as in fig. 3. On the opposite part of the periphery of the same vessel-wall, however, at the same level, that portion of the wall being, so to speak, free to contract or dilate as occasion offered, we generally found a regular layer of large muscle cells, as in fig. 4, as if the very act of continually repeated and interrupted movements had the effect of transforming or developing the branched cells into long fusiform or ribbon-shaped muscle cells or fibres, just as a piece of crystalline malleable iron becomes fibrous by rolling it into bars or drawing it into wires.

In agreement with the foregoing variable conditions in the structure of the walls of veins and lymphatics, we find different arrangements of the lymphatics within the walls. As a general rule, offering few exceptions, the lymphatics do not appear on the inner surface of a muscular layer, when such exists in the walls of veins and lymphatics. We have examined several scores of preparations of the intima, or inner surface of arteries, veins, and lymphatics possessing muscular coats, and in no case have we been able to detect lymphatic vessels there, even when we could see that they existed in great numbers within the gelatinous matrix on the outer side of the muscular coat in the same locality. When, however, no regular muscular layer exists

in the wall, as in figs. 3 and 5, we may, by treating both surfaces of the wall with silver solutions, demonstrate collecting lymphatics on the inner surface, and efferent valved lymphatics on the outer surface of the same portion of wall; and, in cases where the wall is thin, we may make the connection between the two divisions of collecting and efferent lymphatics evident. In other cases, where the muscular layer is a thin one, or formed of one thickness of muscle cells, the silver solution may be made to penetrate through that thin layer from the inner surface, so as to make the collecting lymphatics on its outer surface perfectly evident, as in fig. 4, thus supplying the connecting link between non-muscular veins and lymphatic trunks with collecting lymphatics on the inner surface of their walls and the intima of muscular-walled arteries where the collecting lymphatics never appear, and showing both conditions to be consistent with one general plan or rule.

Let us now trace the course of the intrinsic lymphatics, from the point at which they leave the walls of the various vessels, and consequently in a direction contrary to the course of the lymph stream. In most cases, where the vessel, whatever its nature, is external to an organ, the efferent lymphatic or lymphatics of its wall pass off at right angles to that wall, and there join together to form main lymphatics running parallel to the vessel. In the thoracic duct, the intrinsic lymphatics of its wall bear the same relation to it that the blood-vessels of the walls of arteries and veins do to these structures. In other words, the lymphatics of the wall of the thoracic duct are not branches of the duct, and do not open directly into it, as far as we have been able to ascertain, but join lymphatic trunks passing away from that neighbourhood, but which may afterwards join it near its termination, either directly or through the medium of other large trunks taking that course.

In the case of the intrinsic lymphatics of the walls of arteries and veins of solid organs, like the spleen, kidney, lung, &c., the efferent lymphatics form a plexus on the outer surface of the walls, which lies more or less longitudinally along the wall, allowing the lymph and blood streams to run parallel to each other until the blood-vessels lie free of the solid organ, and the lymphatics of their walls are thus enabled to pass off from them

and join large lymphatic trunks. In the case of the larger vessels, like the aorta and carotid arteries, the vena cava and jugular veins, the plexus of valved efferent lymphatics is dense and complicated, as shown in figs. 1 and 2. In such vessels the efferent lymphatics lie, as a rule, at right angles to the axis of the vessel, within the outer portion of the outer layer or matrix of gelatinous tissue, and forming frequent anastomoses with each other by means of branches showing great irregularity in size; and when they reach that point in the periphery where they are to leave the wall, they pass at once through the adventitia to join the larger lymphatic trunks which lead the lymph away from that neighbourhood.

When we come to the medium-sized arteries and veins, we no longer find the profuse and intricate plexus of efferent lymphatics seen upon the walls of the larger vessels. One, or at most two efferent lymphatics form part of a group with a nutrient artery, and probably a couple of companion veins, and sometimes a nerve bundle. Such groups are found at intervals along the vessels, with a long space between the groups. In such cases the nutrient artery generally lies in the centre, with a companion vein on each side of it, and external to each vein an efferent lymphatic is found, all lying parallel to each other. Upon each efferent lymphatic a large number of valvular dilatations are seen, close to each other, along the course of the lymphatic. From each of these valvular dilatations a straight valveless collecting lymphatic passes off at right angles to the main efferent lymphatic, away from the group, to meet half-way with the collecting lymphatics belonging to another group, and to form typical gridiron plexuses of collecting lymphatics with these, either external to the muscular layer of the arterial or venous wall or, when the latter possesses no muscular coat, to form a plexus immediately under the endothelium of the inner surface, as in fig. 6. The collecting lymphatics, as they pass off at right angles to the efferent lymphatics, lie, of course, parallel to each other, a disposition similar to that often observed in the lymphatics of striated muscle; where, however, the main efferent lymphatics generally lie across the line of the muscle fibres, and the main collecting lymphatics parallel to these fibres and to each other.<sup>1</sup>

<sup>1</sup> "Lymphatiques des Muscles Striés," fig. 1, *Journal de l'Anatomie*, Nov. 1879.

The intrinsic efferent lymphatics generally pass off from the wall of the vessel at the point, or side, which is attached to other structures. Thus the efferent lymphatics of the walls of the aorta, vena azygos, and thoracic duct, leave those vessels where they are attached to the vertebral column.

Returning again to the collecting lymphatics, we have already seen that their ultimate relative position on the walls of veins and lymphatic vessels depends on whether these vessels have a layer of muscles or not in their walls. When the muscular layer is present the collecting lymphatics lie external to it, and do not appear on the inner surface, or intima, as it is called. But, when the muscular layer does not exist, the lymphatics may ramify immediately underneath the lining endothelium of the wall. It often happens that only one long, solitary, valveless lymphatic would thus be seen upon the inner surface of the vessel. At another time a large number of such long, solitary, collecting, valveless lymphatics may be seen, at short distances from each other, on the greater portion of the internal surface of the vessel wall. At other times, and especially when the vascular wall was attached to a firm support, a dense gridiron plexus is seen, of which, the plexus shown in fig. 6, from the inner surface of the wall of the vena azygos, where it was attached to the vertebral column, is an example. It is the most dense and complex of the plexuses we have yet met with.

Having now described the appearance and relationships of the intrinsic lymphatics within the vascular walls, we reach the more important question of their function there. Any attempt to solve that question involves an inquiry into the functions hitherto seriously attributed to the lymphatic system, and into the grounds upon which the belief in such functions rests. We say seriously, in the sense that such functions are believed in by investigators themselves, without referring to the hundred and one supposed functions that are advanced continually by theorists in the current medical literature of the day. That the lymphatics have the function of being peripheral drainage adjuncts, we have always insisted on in our previous researches into the lymphatic system, and this is as plainly demonstrated by their anatomical position and relations as any-

thing could well be demonstrated. We have only, however, looked upon this as the one special function of the lymphatic system; and, as far as the walls of arteries, or the walls of veins and lymphatics possessing muscular coats, are concerned, it may be the chief, if not the only, function of the lymphatics found there, to act as drainage adjuncts. On the inner surface of veins and lymphatics, such a function would be out of place, and their presence there must certainly subserve some other purpose than drainage, for there they carry the lymph away from the interior of the vessel, instead of pouring it into it. The only other generally alleged function of the lymphatics is that of specific absorption, and we have therefore to inquire what grounds there may be for attributing such supposed function to the lymphatics, so far indeed as to give them the name of absorbents, a name given on merely hypothetical and mostly erroneous grounds, but often used in preference to the name lymphatic, founded upon the fact that they contain what is called lymph. Before, therefore, we can ascribe to the lymphatics found upon the inner surface of veins and lymphatic trunks the function of absorption, we must inquire what is meant by the term, and how, and for what purpose, it can be performed in such localities.

It is no exaggeration, we believe, to say that thousands of experiments have been performed upon living animals, most of them of a very cruel and painful nature, for the sole purpose of elucidating this one function of absorption, in relation to the lymphatic system. We cannot call to mind that any other function for the lymphatics has ever had victims devoted to it, but they have all been sacrificed to the demonstration of absorption. And yet nothing could be more unsatisfactory, more contradictory, and more worthless, than the results obtained from such experiments. When we come to inquire what they have proved regarding absorption, we find no clear evidence whatever of the vital phenomena, or power of selection, that is generally understood by the term; but almost all experiments have turned on the question, whether absorption is performed by the lymphatics only, or whether it has been shared in or wholly performed by the veins. These two points have been the foundation of the opposite views of two different schools,—

the first, that of Hunter, the second, that of Majendie,—which have wrangled and experimented without stint over these two divisions in the one conception of absorption. Even in the one locality, and in the one division of the lymphatic system in which the function of absorption appears to be clearest, namely, in the lacteals of the intestinal canal, later views and physical experiments point to a process of dialysis to which digestion is merely an indispensable prelude, forming the food into dialysable materials, a view that almost dispenses with a specific function of absorption. Similarly, the presence of fat molecules, blackened by osmic acid, which may be demonstrated between the epithelium covering the villi, is no proof of absorption, for the same blackened fat molecules can be equally well demonstrated between the epithelial cells lining the sweat ducts.

It was upon the intestinal canal and its lymphatics, or lacteals, that Dr. William Hunter and his school performed their experiments, to prove that only the lymphatics, and not the veins, carried away, or absorbed, nutritive lymph, or chyle, from the contents of the intestinal canal.<sup>1</sup> The plan followed by them was first to choose a very patient (*sic*) animal. They first tried dogs, but these were not patient enough, and anæsthetics were unknown. Sheep were found to be more patient, and more satisfactory; but, as stated by John Hunter, the ass was found “to be patient in the greatest degree, and not so large, nor so strong, but that he may be managed.” The animal was thrown upon his back in a garden, and his legs tied to four stakes. The abdomen was laid open widely, and a portion of the intestinal canal included between two ligatures. Certain coloured and odoriferous fluids were then injected into the included portion of the canal, and without going more minutely into the various steps, it may be said that the fluids referred to were only found by those experimenters within the lacteals, and never within the veins; hence the Hunters came to the conclusion that absorption was performed only by the lacteals. This was the sole result obtained, but upon that result was built up the most astounding hypotheses, many of which still exist, at the present day, without one iota of actual proof.

It may be observed here that subsequent experimenters,

<sup>1</sup> John Hunter's *Works*, by Palmer, London, 1835.



in performing the same experiments, came to the opposite conclusion regarding absorption by the veins; but, for the present, we adhere to Hunter's results, in order to show where they led him and succeeding physiologists in this country.

We have already referred to the experiment so often repeated by Majendie,<sup>1</sup> in which, after carefully dissecting out, and protecting, the femoral artery and vein in the thigh of a dog, the whole of the tissues and lymphatics of the thigh were cut through, and a poison injected into the severed limb. As the poison soon after killed the animal, it was clear that it must have returned into the system from the severed limb by the veins; "therefore," said Majendie, "this proves that absorption takes place by the veins." It was objected, hypothetically, at the time, that there might be lymphatics within the coats of the blood-vessels themselves, according to Hunter's doctrine, through which absorption might be carried on, and that through such channels the poison had passed which killed the animal. Majendie at once met this by severing artery and vein, and connecting them by a quill, through which the circulation was re-established. Still, however, the result of injection of poison was as before. This is supposed to have established Majendie's proposition, although, in this country, it has often been pointed out that the process of injection necessarily ruptured many blood-vessels, that there was therefore virtually injection into the blood-vessels, and not absorption. Thus, the value of this horrible experiment was simply *nil*.

Another experiment of Majendie's, for the same purpose, has hitherto been considered unanswerable, although the present research at once gives a key to it. He carefully dissected out the jugular vein in a living animal, freeing the vein, for a considerable distance, from the surrounding tissue. He then lifted up the vein and inserted a broad piece of cardboard between it and its former bed. Upon the denuded vein, near the middle of the cardboard, he poured a poisonous solution, which touched no other tissue than the venous wall, and yet the animal died. But if any one would only look at the plexus of lymphatics which lies upon, or within, the wall of the vein, shown in fig. 2,

<sup>1</sup> Majendie, *Précis Élémentaire de Physiologie*, Paris, 1836.

and consider that so many lie within a portion only two millimetres square, there would be little difficulty in proving that Majendie's experiment was worthless as far as the demonstration of non-lymphatic absorption is concerned.

These experiments of Majendie's directly involved the question of the existence of lymphatics within the walls of the veins, and were intended physiologically to overthrow the speculations of Hunter, which had led to his conception of lymphatics within vascular walls; and on that account they have an interesting connection with the subject of the present paper. Of course, as far as the results of the experiments made by either Hunter or Majendie, or by their respective followers, were concerned, the absorption they demonstrated was only a modification of the function of drainage, which we have so often insisted upon.<sup>1</sup> It was a draining off of the chyle from the contents of the intestine that Hunter proved to take place by the lacteals; and Majendie only sought to prove a draining away by the veins of the poisonous fluid which he injected or poured upon the tissue or vein wall. So far, therefore, as the opinions of either school are concerned, they throw no light on the use of the lymphatics in vascular walls, the function of drainage having been already discussed by us as inapplicable, at all events, to the inner surfaces of the walls of lymphatics and veins.

Absorption, as conceived by Hunter, was a very different thing from the absorption which is only a kind of drainage; and the question that next arises is whether the lymphatics of vascular walls can have any relation to the function of absorption as conceived by Hunter, and still adhered to as a doctrine at the present day. The absorption he referred to was a solvent and modelling function, which first seemed to bring solid substances into a soluble form, under which form they were subsequently removed by the lymphatics; and it was in this extreme sense that he applied the term absorbents to the lymphatic system. These vessels, according to him, were the prime agents

<sup>1</sup> It is possible that the results obtained by both parties were correct, due we might suggest to the different specific density of the fluids employed by either side. If the poisonous solution employed by Majendie was less dense than the serum of the blood within the vein, it would pass into the vein by endosmosis; if denser, the blood serum would pass out into the solution.

in the absorption of fat, blood-clot, normal and pathological tissues in general, such as tumours, and even bone itself, either in the necrosed condition or in the alveoli of the jaw whence the teeth had been extracted. The difficulty that presents itself to us in respect of looking for any such absorbent function for the lymphatics of vascular walls is, that we consider that all these examples of absorption we have mentioned, as instanced by Hunter, have no foundation in fact in any one case; and even although we find an aortic aneurism causing so-called absorption of the sternum, or bodies of the vertebræ, we cannot admit for a moment that that has anything to do with the lymphatic system, notwithstanding that the lymphatics are numerous on the healthy aorta. To explain this, let us enumerate some of the examples mentioned by Hunter. The argument used is somewhat as follows:—1. He found that liquids in the intestinal canal were taken into the system by the lacteals only; therefore, he says, the lacteals only, and not the veins, are absorbents. 2. The systemic lymphatics are similar to the lacteals; therefore the whole of the lymphatics are absorbents. 3. All fat, blood-clot, bone, or other normal and pathological tissues which *disappear* from any locality are *absorbed*; and as the lymphatics are the only absorbents, they must have absorbed those tissues. 4. As the substances to be absorbed are found everywhere in the body, therefore the lymphatics must exist in every tissue. 5. (Which relates specially to this research). As blood-clot in a ligatured artery becomes absorbed, the intima, or inner surface of arteries, must be well supplied with absorbents, that is, lymphatics. 6. The lacteals appear to communicate with the cavity of the intestinal canal by means of little mouths (the goblet cells); therefore the lymphatics open by mouths upon all the tissues, and especially upon the serous surfaces of the body, including among these the intima of vascular walls.

It would unduly lengthen out this paper were we to show how each of the tissues mentioned by Hunter becomes absorbed or disappears, by virtue of agencies altogether independent of the lymphatic system. In this sense we have our answer ready for each tissue; but for the present it may be sufficient if we consider only the case of blood-clot, as being entirely pertinent to the present research.

In blood-clot absorption the lymphatics can have no share. No one can imagine that there are any lymphatics within the clot itself, whatever there may be in the tissues surrounding it. In the intima of arteries we have shown that no lymphatics exist, so that when blood-clot becomes absorbed from within the occluded ends of a ligatured artery, it is manifestly impossible that the lymphatics can have had anything to do with it. Where blood has become effused accidentally within the tissues, we have been able to follow the whole process of so-called absorption, or rather of organisation, in its various stages, step by step.<sup>1</sup> We have observed that, some short time after the blood has been effused into the tissues, the wandering cells leave the interior of the clot, and subsequently begin to act upon the clot at its periphery, weaving a sort of feltwork of fibres, forming blood-vessels, &c., parallel to that periphery, until the whole clot is organised. At an early stage the red blood-corpuscles break up, forming an amorphous débris, which appears to be eaten, and thus removed by the wandering cells, for the pigment granules can be observed within them. In the whole of this process of absorption, or organisation, no lymphatics are present, and they could thus take no share in the process.

Granting, therefore, that the Hunterian doctrine of absorption can no longer be assumed for the lymphatics, what function can the lymphatics of the inner surface of veins and lymphatics subserve if neither drainage nor absorption can be shown to be necessary or applicable? We are inclined to suppose that the lymphatics here, as elsewhere, have a large share in the reparative, or healing process, after wounds of the tissues in which they are found, by specially supplying the lymph, the pure plastic material by which wounds unite by first intention. The tissues that are best supplied with lymphatics might therefore naturally be supposed to be the ones in which the healing process most readily takes place. The mere presence of blood in a wound seems to be no help whatever to the healing process, for the red corpuscles ought really to be regarded as foreign bodies, which must themselves be removed, as they are removed from organising blood-clot effused into the tissues, a tedious operation

<sup>1</sup> "Zur pathologischen Histologie der Schmerzhaften subcutanen Geschwulst," Virchow's *Archiv*, 4th February 1881, p. 237.

which must greatly complicate the healing process. It is possible that it is on this account that, after a cut of the integument, we find bleeding getting gradually lessened, as if by contraction of the blood capillaries, while finally it stops altogether, and serum, or lymph alone (free from corpuscles), comes from the lymphatics, forms the scab, and heals the wound by first intention. This same hypothesis would explain why in amputation of limbs a skin flap, in which lymphatics are exceedingly plentiful, is of greater value when used without the muscles, in the interior of which lymphatics are rare, although blood capillaries are plentiful. For the same reason, the mesial line or *linea semilunaris*, being (as we have shown elsewhere)<sup>1</sup> exceedingly well supplied with lymphatics, as compared with other portions of the abdominal wall, would be the line of election for making incisions to lay open the abdominal cavity. White fibrous tissue, it is generally agreed, heals more quickly perhaps than any other tissue in the body, and certainly no other tissue is better supplied with lymphatics, Professor Sappey's opinion notwithstanding.

The hypothesis which we have just stated, in as far as it is applicable to the blood-vessels, receives a large amount of corroboration from some experimental researches undertaken many years ago by the late Professor Spence, and which therefore at the present time may have here a special interest for the Edinburgh School of Medicine. Professor Spence tied a number of arteries in dogs, which were killed at various intervals after the operation. These tied vessels were afterwards examined, and they showed conditions not only entirely consistent with the anatomical facts which we have described in this paper, but which might have been surely predicted from these facts, if the knowledge of them had preceded the experiments. The experiments were made for the purpose of investigating the changes which take place in the arteries of the human body which have been ligatured for aneurisms; and in order to avoid even the suspicion of warping the sense to suit our own hypothesis, to which a description of the results of these experiments in our own words might be liable, we shall quote *verbatim* a paragraph on this subject from the pen of the late Professor Miller, as

<sup>1</sup> "Lymphatiques des Muscles Striés," *loc. cit.*

found on page 543 of the third edition of his *Principles of Surgery*. Professor Miller says of such ligatured vessels :—

“Plastic exudation is not limited to within the vessel. It occurs, and more extensively, on its exterior, forming a dense swelling of some size, within which the ligature’s noose is deeply embedded. The highly important, and even essential character, of this external exudation of plastic fibrin is fully elucidated by the experiments of Mr. Spence, from whose unpublished drawings the illustrations herewith given are derived. He has shown clearly that the internal clot, so far from being, as was supposed by Manec and others, essential to the separation of the ligature without hæmorrhage, is not unfrequently wanting when the vessel has been successfully tied, the closure being entirely effected in these cases by the plastic exudation between the cut edges of the internal coats and by the bulky fibrinous mass within and around the sheath. It is this external exudation which, becoming vascularised, forms the medium whereby blood is supplied from all the surrounding parts to the important new formations within the external coat of the occluded vessel; and which, by its equable pressure upon the divided ends of the internal coats, prevents the tender adhesions within the sheath from being broken up, even when there is no coagulum. According to Mr. Spence, the vessels of the new plastic lymph are formed with great rapidity. In one instance, in the dog, he found them present in considerable numbers sixty hours after the operation. After a longer time, vessels begin to pass even into the clot when this is present; these vessels being always in connection with those of the sheaths and external lymph, and not proceeding, as has been supposed, from the interior of the artery.”

Comment on the above description, given for the sake of comparison with the facts which we have discovered, is scarcely necessary. The lymphatics which we have described as ramifying in what is called the outer portion of the middle coat would be completely ruptured in “satisfactory tying,” leading to the effusion of their contained lymph only where it has been described as lying. Indeed, the facts being as we have described them, the consequences could not well be otherwise; and the subsequent direction of vascularisation only tends to confirm

the hypothesis which we have attached to the lymphatics, in addition to their main function of drainage adjuncts. The whole hypothesis is, however, only put forward tentatively by us, as a means of answering the question propounded by the condition of the lymphatics themselves on the inner surface of the walls of veins and lymphatics. It may be wrong, but if found to be so, another function must be suggested that is distinct both from the drainage function or the absorption which is synonymous with it.

The following conclusions form a summary of the results obtained by us :—

1. No lymphatics exist in the intima or on the inner side of the musculature of large arteries. They ramify in great numbers only within the gelatinous layer lying on the outer surface of the musculature between it and the tunica adventitia.

2. The foregoing conclusion holds good also in the case of the walls of large veins and lymphatics possessing muscular coats.

3. In those portions of the walls of large veins and lymphatics in which the muscular coat is absent, the collecting lymphatics may form a gridiron plexus, with few or no valves, almost immediately underneath the lining endothelium.

4. In all three kinds of vessels the intrinsic efferent lymphatics carry away the collected lymph from off the walls, and do not open into the cavity of any of these vessels.

5. While in the walls of vessels possessing muscular coats the lymphatics seem specially adapted as drainage adjuncts for such vessels, they must have a separate function in the cases of veins and lymphatics, where the collecting lymphatics are found immediately underneath the endothelium. That function is probably to aid in the repair of wounds of the walls of these structures.

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## DESCRIPTION OF PLATE I.

*(Drawings made by the aid of the Camera Lucida.)*

Fig. 1. Plexus of intrinsic lymphatics lying on the outer surface of the musculature (?) of the aorta of the horse. The upper portion are collecting, the lower efferent lymphatics ( $\frac{1}{3}$ ).

Fig. 2. Plexus of efferent intrinsic lymphatics lying on the outer surface of the musculature of the axillary vein in the horse ( $\frac{1}{4}$ ).

Fig. 3. A portion, *cl*, of fig. 5, greatly magnified, showing part of a plexus of collecting lymphatics lying immediately underneath the lining endothelium of a non-muscular portion of the wall of the receptaculum chyli in the horse ( $\frac{1}{110}$ ).

Fig. 4. A portion of a plexus of collecting lymphatics lying immediately external to a thin muscular layer in the wall of the thoracic duct in the horse ( $\frac{1}{110}$ ).

Fig. 5. Plexus of collecting lymphatics lying amidst the branched cells, and immediately underneath the lining endothelium of a non-muscular portion of the wall of the receptaculum chyli in the horse; *cl*, track of an efferent lymphatic seen on the outer surface of the wall ( $\frac{1}{10}$ ).

Fig. 6. Gridiron plexus of collecting lymphatics on the inner surface of a non-muscular portion of the wall of the vena azygos, lying immediately underneath the lining endothelium, from the horse ( $\frac{1}{14}$ ).

*a*, artery; *c*, branched cells; *cl*, collecting lymphatics; *el*, efferent lymphatics; *m*, muscle cells.



MICROCOCCLUS POISONING. BY ALEX. OGSTON, M.D.,  
*Surgeon to the Aberdeen Royal Infirmary.*

(Continued from vol. xvi. p. 567).

SAPRÆMIA.

(σαπρός, putrid; αἷμα, blood.)

THIS term was first used by Dr. Matthews Duncan<sup>1</sup> to signify "mere poisoning by the chemical products of putrefaction," and concerning it he says:—

"Sapræmia, or simple putrid intoxication—poisoning not by an organism multiplying in the blood, but by the passing into it of the chemical products of putrid decomposition—is one upon which much light has been recently thrown, and with the most beneficial results in practice. Like the other forms of the so-called puerperal fever, this I shall treat as a separate entity, and it frequently is so. But it may be combined with the traumatic fever of inflammation, and it is especially liable to be combined with septicæmia and pyæmia. Indeed, it has long been, and still is, the habit to speak of septicæmia and pyæmia as diseases of putrefaction, but this is a mistake. Putridity of the discharges is not an essential part of these diseases at all, though it often accompanies them. The organisms which cause septicæmia and pyæmia probably take no part in putrefaction. They live in the discharges, and are conveyed or pass into the blood, where they multiply indefinitely. The organisms which cause putrefaction, whether the *Bacterium termo*, or others in addition, may pass into the blood with the putrid fluids to produce sapræmia, but they do not survive, far less grow therein.

"We have then, in sapræmia, when uncomplicated, a very simple problem. Putrid ichor is absorbed, or flows through the uterine sinuses, or otherwise, into the circulation. Its poisonous constituents are eliminated rapidly from the blood, for if the supply is stopped the sapræmic phenomena quickly disappear.

"When once in the blood it does not increase in it, ferment-like, independently of any other supply. Sapræmia is kept up by a continuous supply of the poison. It disappears when the supply from without is stopped. To stop the supply is the problem of the cure."

In all that concerns sapræmia, this description is probably perfect. It is beyond doubt that there are organisms (saprophytes) that produce *common putridity*, and others that cause

<sup>1</sup> "Puerperal Fever," *Lancet*, 1880, vol. ii. p. 684.

*decompositions that are not putridity.* To the former belong most, if not all, forms of bacterium, some bacilli, and some spirilla. To the latter belong some bacilli and some micrococci. If micrococcus be introduced into a fluid and there cultivated, it produces no offensive stench; its ptomaines are not those of putridity. In most acute abscesses, where it is found in abundance, no bad odour is detectable,<sup>1</sup> and if the contents of an abscess, or a fluid where organisms have been growing, be found to present a putrid smell, we are absolutely certain to detect in it numerous organisms that possess either the sausage shape of the bacterium or the rod form of the bacillus.

These sapro-micro-organisms produce disease in a manner peculiarly their own. Koch<sup>2</sup> thus describes their mode of action :—

“Five drops are enough to kill a mouse within a short time. Immediately after they have been injected, distinct morbid symptoms are observable in the animal. It becomes restless and runs much about, yet showing great weakness and unsteadiness of movement, ceases to eat, the respiration becomes irregular and slow, and death ensues in four to eight hours. In such animals the connective tissue of the injection site still contains the greater part of the injected fluid in the same condition as when it was injected.

“It shows the same numbers of various organisms mixed through each other, just as the microscope showed prior to injection; but there is no reaction in the neighbourhood, the viscera are unaltered, the blood taken from the right auricle produces no result if inoculated on another mouse, and organisms are found neither in the blood nor the internal organs.”

The animal has been poisoned by the ptomaines—*i.e.*, the gases and liquids of the sapro-micro-organisms.

In man we find this sapræmia in various forms and degrees. The slightest form is the sickness and nausea produced by a bad smell, which is but a ptomaine of putridity, and which has been known to produce serious symptoms without the possibility of entrance into the body of the micro-organisms that generated it.

Its usual and most marked manifestations are in the sapræmia of childbed, which Matthews Duncan has described, and in

<sup>1</sup> My endeavours to ascertain the kind and properties of the ptomaines of micrococcus have hitherto failed.

<sup>2</sup> *Wundinfektionskrankheiten*, p. 40.

foetid abscesses such as those in or near the abdominal cavity—in the former case it may exist alone, in the latter its symptoms are mixed with those of septicæmia.

Where pure, as after childbed, its symptoms are the rising temperature, the hot skin striving by perspiration to throw off something that the system is resenting, the full pulse, the flushed face, the headache, and the occasional tendency to gastro-intestinal derangements. We see, in fact, that the system is poisoned, and the foetid lochiæ, with probably putrefying clots, are evidently the pool of poison that is causing the evil. We wash out vagina and uterus, or it may be that we merely set our patient erect for a few moments and allow the foetid clots and contents of the vagina and uterus to escape, and our disease is gone, defevrescence is the work of but a few hours, and the patient is cured. It is a poisoning, not an infection. In the blood we find a few organisms of the bacterium shape, the urine shows a number of long organisms with the characters of bacillus, but anything like a blood disease does not exist, there is but an intoxication.

Where complicated with septicæmia, as in most cases of foetid abscess, the symptoms are a mixture of the two diseases, but the great alleviation, the improved colour, the diminished perspiration, the relief of the aching or oppressed head, and the return of the appetite that follow the issue of the stinking pus, point to the sapræmia induced by it being a condition *per se*, and the patient seems on the way to convalescence and health when it has been removed. Very often, however, the symptoms of septicæmia, that were little noticed in the presence of those of sapræmia, gradually come to the front, the improvement may be but temporary, and the patient may die after all.

In sapræmia, whether natural or produced experimentally, the sapro-micro-organisms that enter the blood are evidently too weak to overcome its vitality, for they do not show much evidence of being alive; they do not survive and cause putrefaction in the cavities or fluids, normal or pathological, into which it may convey them; they are probably consumed by the leucocytes, or in some other manner, for the urine in this disease, although it usually contains forms of bacilli alive and growing, that have passed into the system coincidently with the sapro-micro-organ-

isms, shows no trace of putrefaction nor of the forms of bacteria that produce it.

The sapro-micro-organisms are probably enfeebled or killed through their removal from the oxygen of the air, which is necessary for the full exhibition of their decomposing energies. I have already pointed out that bacteria grow best on the surfaces of liquids, where they form thick turfs in contact with the air, while at the bottom of the fluid, in deep jars, they are either dead or comparatively inactive. In such a state they are found in the blood in *sapræmia*, and are doubtless killed where the individual is in any save the most enfeebled condition, although the early onset of decomposition, commencing ere life is extinct, that we find in those who die from septic processes, as well as the fact that when they are injected into a very weakly and ailing animal, such as one dying of diarrhœa or other enfeebling disease, they may be found alive and growing in the blood at the moment of death, point to the probability of the entrance into and growth within the system of sapro-micro-organisms even before life has become extinct in all forms of fatal enfeebling disease.

#### SEPTICÆMIA, PYÆMIA, AND SEPTICO-PYÆMIA.

It will conduce to clearness and brevity at once to state that every fact that I have been able to observe regarding these diseases points to their being one and the same, and to their sole and invariable cause being *micrococcus poisoning*.

*Micrococcus* is met with in two distinct forms, chains and groups. They are often found together, yet the two are different, and the chain form does not pass into the grouped form, nor the grouped into the chain form. Throughout this paper the term *micrococcus* is used as embracing both forms; the chain coccus is often called *streptococcus* (Billroth), and I shall call the grouped form *staphylococcus* (from *σταφυλή*, a bunch of grapes).

*Micrococcus*, which, when limited in its extent and activity, causes acute suppurative inflammation (phlegmon), produces, when more extensive and intense in its action on the human system, the most virulent forms of septicæmia and pyæmia, as well as many forms intermediate between the two extremes.

The connection between suppuration and blood-poisoning is

no new idea; the very name *pyæmia* was conferred as implying the relationship; and the title of "purulent infection," though little used in English or German literature, is still the favoured term for the disease among the French clinicians. Nor is the idea of septicopyæmia being equivalent to micrococcus-poisoning new, it has constantly been cropping up in various forms.<sup>1</sup>

Although at first it be somewhat repugnant to existing notions to consider acute inflammation but a slighter form of pyæmia, very little reflection will show that the statement is by no means a strange one. The common acute abscesses with which we are all so familiar are attended with febrile conditions not very unlike the fever of blood-poisoning, and the circumstances under which they occur, such, for instance, as in women shortly after childbed, are the very times when we also meet with the graver disease. Not only so, but acute inflammation, such as pneumonia, are very apt to end in blood-poisoning, and in the old and enfeebled, where this is naturally most usual, a separate clinical form where this is the rule has been described and rendered classical by Trousseau.<sup>2</sup> Acute inflammations of the peritoneum are closely connected with septicæmia, and acute suppurative inflammations of joints, as in urethral rheumatism, bring us a step nearer to pure blood-poisoning. Between an acute inflammation (whitlow) and a poisoned wound of the hand, there is no possibility of drawing a valid distinction, for not only do intermediate forms occur, but the one usually or frequently passes into the other. There is in reality no distinction between the erysipelatous blush surrounding an acute abscess in its angriest stage, the erysipelatoid infiltration that is common around the wounds of the septicæmic, and the phlegmonous erysipelas that is so often fatal from blood-poisoning. Acute suppurative inflammations of the arachnoid and pleura never occur save as part of a process of blood-poisoning; a suppurating ear needs but to extend itself to the bone and the lateral sinus (or brain), and, with no other change than that of site, has become a fatal septicæmic malady. Suppurations under the

<sup>1</sup> *Cocco-bacteria septica*, by Billroth, 1874; also *Cocco-bacteria septica*, by Billroth and Ehrlich, 1877, *Arch. f. Klin. Chir.*, vol. xx. p. 403; and elsewhere.

<sup>2</sup> *Clinique Médicale*, ed. 2, vol. i. p. 747.

scalp and in the diploë are dreaded chiefly owing to their intimate connection with septicæmic conditions; parotid abscess and acute suppurations in the glands of the neck are also almost synonymous with pyæmia. A quinsy runs its course with feverish symptoms that are of the same nature; necrosis of the jaws is always accompanied by septicæmia, as are also all acute suppurations of the thyroid gland. All acute abscesses about the thorax and abdomen, such as hepatic abscess, abscesses of the abdominal wall and around the cæcum, are of the septicæmic type; splenic and hepatic abscesses, renal abscesses, and miliary abscesses in these viscera and the heart, are all common conditions in pyæmia. Abscesses in the glands of the extremities, and among their soft parts, are always connected with septic or erysipelatous processes; while to name acute suppurative inflammations in bones or joints is but to name maladies whose whole appearance is that of blood-poisoning. It would be hard to find an acute suppuration anywhere where general symptoms of blood-poisoning may not be a prominent feature.

Since at all hands evidence is abundant that acute suppurative inflammation is closely allied to blood-poisoning, so closely that in any given case we have only to imagine it indefinitely increased to see that it must eventuate in the latter disease, the fact that nature shows so great a difference between the milder and the graver forms must not be allowed to blind us into the rejection of what can, I think, be shown to be an indubitable truth, and capable of being in all points rationally explained. There is nothing surprising or unexampled in mild cases of disease of the bacteric (zymotic) type, being very unlike their severe forms, and as examples may be quoted the instances of a mild attack of typhoid fever where a little feverishness and diarrhœa is almost all that is observed, and the virulent forms of the same where the patient may be even struck down comatose, like one in apoplexy, and die in stupor ere any of the usual appearances occur; or a mild attack of vesicular smallpox, as compared with a bad hæmorrhagic case where death occurs ere the eruption appears. It must be admitted that the difference between acute suppurative inflammation and septicæmia is not an unparalleled phenomenon, if it be the case that they are forms of one and the same disease.

Already, in an earlier part of this report, the theory has been developed that what we call inflammation is but a form of reaction, a perversion of the process of growth, which the tissues and the structures of the body present when injured by anything capable of damaging them, each irritating cause having its own specific reaction recognisable by the form of inflammation it presents, and that the characteristic reaction of micrococcus is acute suppurative inflammation (phlegmon).

I have also suggested that the term septicæmia is a misleading one, as in no case is the blood the focus of disease, but that this is to be found in the tissues, whence the ptomaines of the disease pass into the circulation to act as poisons or intoxicants, and separate individuals or small groups of the micro-organisms are conveyed by this fluid into other situations so as to reproduce among other tissues the disease of the parent focus. The term micrococcus poisoning, which does not include the idea of an essential blood-disease, would be in some respects a more suitable name than pyæmia or septicæmia. But to designate the more severe forms of micrococcus poisoning, and the conditions in which we infer from the symptoms that the system generally is becoming poisoned, I shall still retain the term septicæmia as a name, although refusing to agree with the belief that the condition implied by it really is an infection of the blood *per se*. Where the term septicæmia would mislead, the word "ptomaine-poisoning" will be made use of.

#### PHLEGMON OR ACUTE SUPPURATIVE INFLAMMATION.

The most ready means of studying this condition is to observe the results of injecting, into the tissues of animals, micrococcus as it exists free from admixture of any other micro-organism, in the pus of an acute abscess. Although micrococcus is perhaps the greatest scourge that afflicts the human race, it is by no means so pernicious to animals when injected, and some species are considerably less easily affected by it than others. Guinea-pigs are, I think, decidedly less sensitive than mice.

If two minims of the pus be injected under the skin of a mouse's back, the animal, on awakening from the chloroform, withdraws into its nest, where it remains for a few hours in a state of listlessness, probably due to the anæsthetic. It makes

its usual journeys to its food, and appears in a fair way to recovery, when, after the lapse of five or six hours, there commences a condition, well described by Koch, and characteristic of blood-poisoning. It leaves its nest, sits in the open air in a corner of its cage, its hair is disordered, its eyes glued together, it neither eats nor drinks, nor does it attend to sounds or impressions that used formerly to attract or terrify it.

This condition may end in several ways. It frequently occurs that at the site of injection, at the end of a day or two, a hard thickening is felt, extending and becoming more elevated, until fluctuation can be detected at its centre, and the process results in the formation of an acute abscess. When this occurs the general symptoms become less severe in proportion as the local symptoms grow more marked, so that, by the end of five or seven days, the former have generally disappeared, and the animal is again eating, and as lively and active as ever, although it bears on its body a small well-marked abscess at the spot where the micrococci had been injected.

If the animal be killed during the first few days, when the general symptoms are well-marked, there is found locally a red knot with a yellowish spot in its centre, and when the knot is bisected the hard red circumference and yellow centre present a picture very like that which we observe during the spreading of a soft chancre. The centre seems to be softening or liquefying into purulent matter, and the margin is thick and infiltrated around the liquefying edge. A cross section of the edge shows the presence there of dense clouds of micrococci, which, when the ordinary grouped form of coccus (*staphylococcus*) has been injected, are working their work of invasion in rounded masses, like clouds of dense smoke, that can when properly tinted be seen to be swallowing up the tissues by uniform peripheral invasion, the tissues a little way beyond them being rendered waxy and homogeneous, so that cell, nucleus, and intercellular substance are no longer so distinct as usual; and this halo of altered tissue apparently the result of the irritant caustic products of growth acts as an advanced guard to the clouds of *staphylococci* that follow and destroy all semblance of structure in the tissues, ere the latter finally deliquesce in their rear into a purulent fluid.



If the coccus injected has been the chain form (streptococcus) the process is a little different. There is a similar central breaking down into pus, perhaps not so rapid in its advance, and hence, resembling less the destructive work seen in a soft chancre; there is a like waxiness of the tissues invaded, but the invasion is not by dense clouds that destroy all trace of structure, but by the insinuation among the tissues of chains of cocci that pervade the intercellular substance and cells, forming a network of lines, between which may be seen the nuclei of the tissues. The invasion is more like the enamel on "crackle ware" china, present everywhere, yet permitting the pattern of the tissues to remain distinct.

At some distance around these foci of micrococcus growth the tissues are at first little altered, but as time goes on they show more and more condensation and thickening, the elongated cells tend to become round and the fibrous intercellular substance gelatinous, the tissues pass in fact into embryonic connective tissue or granulation tissue, and a layer of this comes in time to surround the knot.

Before it has formed the animal suffers from septicæmic symptoms, and in its blood everywhere, usually to be found without much difficulty, and in nearly every slide of blood, exist numbers of micrococci, single, in pairs, or in a small group or short chain of three or four, floating in the serum of the blood, and presenting no special affinity for the blood globules, either red or white. Now and then a group of micrococci of not inconsiderable size, containing as many as perhaps fifty individuals, is to be met with, but this is rare. I have seen it only once.

The eyelids furnish during this stage a purulent discharge containing micro-organisms, and among them numerous micrococci.

As the granulation wall around the abscess (which is evidently a reactive process by which nature, in a strong animal, limits what would otherwise be an indefinite disease) begins to form, the general symptoms tend to subside, as if it impeded the passage into the system of the micro-organisms and their ptomaines. When once the wall is strong and thick, the entrance of the micrococci is arrested, the abscess is as it were

sequestered from the system, the septicæmic symptoms disappear as already described, and the occurrence of a micro-organism in the blood becomes a matter of greater and greater rarity.

But it does not invariably occur that *abscess* results. In some animals that seem to possess unusual immunity or powers of resistance, or where the dose has been a small one, the blood-poisoning symptoms begin to pass off in a day or two, and the local induration and infiltration never reach the stage of supuration, but fade and disappear, ending in *resolution* without any further evil occurring. In some cases, on the other hand, where the reverse conditions of susceptibility or dose obtain, more severe effects are produced, as will be afterwards explained.

It will be seen, in the former report, that no such results follow the use of chronic pus, which contains no organisms, nor of pus where these have been destroyed by heat or carbolic acid; while on the other hand the micrococci, if removed from the pus and suitably cultivated, produce the same results.

This process of acute suppurative inflammation or phlegmon finds many an identity in man. Without dwelling on such a disease as soft chancre, which is an ulcer full of micrococci and other germs, but whose specific organism must be cultivated under conditions so peculiar that the disease deserves a separate description and a separate study, we find in such illnesses as poisoned wounds of the fingers and hands, so common among the working classes, in whitlows, quinsies, mammary abscesses, and common acute abscesses generally, a state of matters exactly corresponding, save that we have to hazard a guess as to the method of inoculation, being unable to demonstrate its when and how. In poisoned wounds of the hands and in quinsies, where the inflamed part communicates directly with surfaces abounding in germs of many forms, we can always suppose, and in many cases more than suppose, the introduction through a breach of surface of some extraneous matter that may convey the micrococci.<sup>1</sup>

<sup>1</sup> It is readily conceivable that in some phlegmons, such as quinsies, a rheumatic or gouty diathesis may primarily cause the inflammation, which micrococci, when ingrafted, may drive on to suppuration. Here the diathesis is the first cause, and the micrococcus the cause of its terminating in suppuration. Micrococci may modify a process begun by another cause, but they also cause inflammation without requiring such process to pre-exist.

We can also, in deeper lying whitlows about the fingers and palm of the hand, arising as they do not in the tendinous sheaths, but in the lymphatics that course in front of them in their passage between the cutaneous lymph spaces and the absorbent system of the arm, infer from their situation and connection with the absorbent system their causation from a surface breach or surface infection that may have been overlooked, or have been so slight as to be undiscoverable. Mammary abscesses and acute pelvic abscesses can be traced to the existence of the puerperal condition, and the favouring source of infection in the uterine and vaginal discharges of childbed that render frequent, at the same epoch, various other diseases whose septicæmic relations are more generally recognised. Common acute abscesses elsewhere can with wonderful frequency be traced to some local source of septicity, or to some disease more or less connected with blood-poisoning, or favouring the circulation of dangerous organisms in the system, such as fevers of various sorts, diphtheria, erysipelas, or ulcerative processes about lungs, bowels, or other viscera.

But if it be difficult to trace the precise mode of inoculation in each individual case, the demonstration of their connection with micrococci offers but little difficulty in their later stages. In each pustule or abscess the evacuation of the liquid enables us, with unfailing certainty, to recognise the presence in it of immense numbers of these bodies, amounting to hundreds of thousands or even millions in each drop, and where it is possible at the same time to scrape away a little tissue from the walls, we find in the early stages the same rich invasion of micrococcus as was described in connection with animals. We have also evidence of their passing into the system, for in many instances, especially about the upper extremity, we can observe, in the implication of the absorbents, in the lymphangitis or lymphadenitis, the inflammatory reaction that shows the absorption of some irritating matters, probably both ptomaines that irritate the walls of the vessels they pass through and micrococci which traverse without lodging in the vessels to be retained in the first lymphatic gland they meet with, where they are filtered out of the blood, and develope to an extent dependent on their virulence and the susceptibility of the individual. When their

growth in the lymph gland goes on to the extent of suppuration, we are in a position to verify their having passed from the original focus into it, by their occurrence in large numbers in the pus which is removed from it.

It often occurs that the process extends no further, that the lymph gland seems to be the limit of the extent to which the disease penetrates the individual, as if the ptomaines and micrococci passed no further, or only in so dilute a form that they neither cause general disturbances nor can be demonstrated in the blood. But this is by no means always the case. It often happens that sufficient of the germs and their products pass the lymph barrier to give rise to ptomaine-poisoning of the whole system, revealed by fever, shivering, rise of temperature and other general disorders, while the micrococci can be found in the blood by examination of a series of slides prepared from it, numerous enough to enable us to investigate a sufficient quantity. Where the fever runs high and the symptoms indicate that absorption is abundant, every slide of blood may prove the presence of micrococci, while in less severe cases several slides, four or six, or it may be ten or twelve, may be gone through ere a micrococcus is found. With diminution of intensity their demonstration becomes more difficult, until in the mildest cases it is well-nigh an impossibility. The number of micrococci in the general circulation is in proportion to the severity of the disease. So also the numbers of micrococci in the pus are in proportion to the virulence and intensity of the inflammation. In acute abscesses of the connective tissues, we are less in a position to trace the exact course by which the micrococci and their ptomaines enter the system generally; but we observe the same fever and general symptoms due to their presence, and they are also to be found present in the blood in a proportion that corresponds accurately enough with the evidences of their generalisation that we can obtain from the signs at our disposal.

The completion of a well-formed demarcating wall of granulation tissue around the abscess coincides as a rule with the disappearance of the general symptoms in the individual and of demonstrable micro-organisms in the blood. Both in man and in animals the production of this wall puts an end to the pro-

gress of the local invasion. The interior of the wall of a ripe abscess presents no clouds of invading cocci, but shows only a few, and these growing but feebly among the cells on the innermost layer of the wall. Their race is run so far as that locality is concerned, and even among the pus of the cavity they no longer multiply with their pristine energy, but become more and more diluted by the liquid that collects under the influence of the tension in the cavity, so that their numbers lessen considerably in relation to the quantity of liquid. The streptococcus, under these circumstances, runs a course similar to the staphylococcus, save that in ripe abscesses due to the former there are frequently found chains truly enormous in length, where the individuals can be counted by several hundreds in a row.

There is another quarter whence we obtain evidence that micrococci are circulating in the system in these cases, and that is from examination of the urine. Normal urine contains no organisms, but in the feverish conditions resulting from phlegmons the urine frequently presents micrococci, often along with bacilli, in masses and groups, indicative of their having grown after escaping from the kidney.

#### ACUTE SUPPURATIVE CATARRH.

It is not so easy to demonstrate the casual connection between catarrhs and micro-organisms, as they necessarily occur in situations where communication with the surface of the body is free, and where therefore organisms *may* normally be found. But in every such catarrh, as gonorrhœa and purulent ophthalmia, the discharge is very rich in various forms of micro-organisms, far richer than could possibly be supposed to be the case had they no connection with the disease, and among these organisms micrococcus holds an important place. It is very abundant from the beginning, and as the disease diminishes in intensity, and the other forms become less copious among the discharge, the epithelial scales that are cast off are inhabited by large numbers of micrococci, as if the disease had been caused by their spreading among the cells forming the lining of the canal. When the disease approaches cure, the inhabited cells diminish in number, but they, as well as micrococci free among

the discharge, persist until the end, and disappear only with complete cure.

I have no experiences to detail regarding purulent catarrhs induced and studied in animals. All the observations made were in man, and these have resulted in the belief that they are due to a superficial dermatitis, of the nature of catarrh of the epithelial layer, from a growth in that layer of micro-organisms, in all probability micrococci. I have made no observations on the blood in these cases, and cannot tell whether it usually contains micro-organisms.

#### SLoughING INFLAMMATION OR INFLAMMATORY MORTIFICATION.

Necrosis of the soft parts from acute inflammation can be induced in animals, and offers an excellent illustration of a disease standing midway between septicæmia and acute inflammation, presenting the features of both.

Owing to the micrococcus in human diseases being less potent in the lower animals than in man, it is not easy, as in abscess, to produce necrosis at will, but it now and then occurs, especially in mice, that among a series of inoculations, either from peculiarities of the animals or the poison introduced, a good example of inflammatory necrosis of the tissues is obtained for study.

In these cases, when a minim or two of pus has been injected, there occurs, instead of the infiltration ending in abscess, a more intense inflammatory process in which the tissues are so damaged in their vitality that they necrose almost at once, and ere they have had time to show the usual phenomena of inflammation and abscess formation. The sloughing occurs within the first two or three days, during which time the animal has shown the usual signs of septicæmia, and its blood presents, if examined, the usual admixture of micrococci. If the necrosed tissues be examined they are found full of stripes, bands, and larger masses of staphylococci, that occupy the soft parts, pervade them everywhere, and seem to affect mostly the connective tissues of the part, although they may also be seen in the lymphatic spaces, forming large spongy tufts in the midst of the coagula by which these vessels are obstructed. But the type of the disease, in the specimens I have examined, has been a *dense*

*infiltration of the tissues by the staphylococcus* in bands and masses resembling somewhat densely veined marble or the clouds in a windy sky.

Animals rarely die with this form, owing, I presume, to their natural resistance to the organism that produces it, so that it has occurred to me to study its fatal issue only in man. In mice the disease generally terminates in a limitation of the sloughing process within the first five days, and in a rapid separation of the necrosed tissues, with disappearance of the general septic condition. The necrosis affects only limited portions, patches of skin and subcutaneous tissue, and does not penetrate deeply, or affect entire limbs. So soon as the necrosis limits itself, the surrounding tissues exert themselves to isolate it by the formation of a wall of granulations, which bring about its detachment, and act, as formerly described, as a hindrance to the passage of the ptomaines or of individual cocci into the system at large, so that the animal soon ceases to present evidence of either of these occurring, and is afebrile and healthy by the time the slough has separated. The ulcer remaining at the spot is speedily filled up by granulation and cicatrisation.

It is in man that this disease is oftenest seen in full perfection, and it presents the following picture in a more or less intense degree. Starting from some cut or scratch that is generally to be distinctly made out, and affecting usually an upper extremity, a spreading inflammation, in which the surface dermatitis is the most evident, but where the deeper parts are also swelled and phlegmonous, so that the disease possesses a certain likeness to erysipelas, extends from its point of origin among the soft parts, binding them into a swollen and brawny mass, over which the epidermis is tight and glistening, or even blistered here and there from tension, showing a series of smallish vesicles. These, at first discrete, speedily flow together into larger patches. Though in less intense forms vesication may not occur, the subsequent desquamation shows that the epidermis has sympathised with the inflammation.

The pain, burning in its nature, at first local, but rapidly extending, and the elevated temperature of the part, raise in the minds of even the laity the suspicion of a poisonous matter having found its way into the limb; and this is heightened by

the involvement of the lymphatic system, shown by pain, tenderness, enlargement and induration in the cubital gland or the absorbent glands of the axilla, accompanied occasionally, but by no means frequently, with tenderness and induration, or even redness, along the line of the lymphatic vessels leading to them.

The penetration of ptomaines into the general system is shown by the vomiting, malaise, and feverishness that are usually present at an early stage.

The process does not present the raised edge of erysipelas.

As the disease advances, œdematous swelling precedes it in its advance up the arm, and before the elbow is reached the parts of the hand earliest invaded have gone on to abscess in the milder, and gangrene (necrosis) in the severer forms.

When necrosis results, it affects entire fingers or the whole hand, which become purple and finally black in colour, the general condition becoming greatly aggravated, with anorexia, foul tongue, sweating, flushed face, hot skin, and symptoms of collapse, the intellect remaining clear until the end.

Should death not occur, extensive suppuration in the limb, with separation of sloughs of the connective structures and of the necrosed fingers or hand, follows; and it is not unusual for the lymphatic glands of axilla or arm to suppurate as well.

Microscopically, this disease is identical with the necrosis just described in animals. The soft parts are invaded with bands and colonies of the staphylococcus scattered everywhere among the connective structures, so as to resemble the markings on marble or on pitch-pine wood. The fluids in the vesicles if opened at an early stage, show no micrococci; they are due to mere tension; but later on they, as in all the liquids of the part, present themselves in great abundance.

The blood of the body shows micrococci isolated, paired, or in very small clusters, and the number found is fairly proportionate to the intensity of the disease. In mild attacks they may be so few as to be discovered with difficulty, but they are easily found where it is severe; and in the urine they are usually present, along with a few other organisms, such as bacteria and bacilli, where the general symptoms are at all pronounced.

This disease, whose dependence on micrococcus poisoning can be followed and demonstrated on parts removed during life as



well as after death, offers one of the best instances I know of the difficulty, or even impossibility, of demonstrating its cause by the older and less perfect methods. By dry lenses and water-immersions I have sometimes observed nothing, in the sections of the soft parts, that could account for its existence. Even by the application of Koch's processes, viz., the methyl-aniline staining, the oil-immersion lenses, and the Abbé's illumination, it has often chanced that nothing was at first seen in the soft parts; so that more than once the investigation was about to be given over in despair, when the use of some other staining, notably of the aniline Bismarck-brown, has revealed the most exquisite and universal groups of staphylococci, as unlike any other structures as could well be conceived, pervading the flesh like mathematically-arrayed fish-roe or the cells of a honey-comb.

The older methods gave negative or contradictory results, while Koch's processes have always *eventually* yielded ample proof. As far as I have tried Weigert's gentian violet in this disease, it has never failed to give brilliant results.

In this disease the tendency to localisation of micrococcus is a marked feature. Although they exist everywhere in the tissues, they are seen only in close-packed colonies separated by intervals of uninvaded tissue, cohering into colonies with great tenacity. They do not transude into the serum of the blisters until a comparatively late stage has been reached, and their behaviour produces the strong belief that, though they poison the system, they do so far more by sending their ptomaines profusely into it, than by infecting it by detached members of their community. Septicæmia, as we are wont to call it, is present, but it is plainly more a process of ptomaine-poisoning than a growth of micro-organisms in the blood.

#### ERYSIPELATOID DISEASE.

Under all forms of wound treatment there appear slight and evanescent forms of erysipelas, concerning which we are in doubt whether they are the same disease as the typical erysipelas *migrans* of the head and face. They commence with chills or rigors, generally associated with vomiting, and the absorbent

glands into which the lymphatics of the invaded part drain are swelled and tender. They often want the spreading margin, and are so evanescent that it rarely happens that the term *migrans* can with propriety be given them. I have had no adequate opportunity of investigating the pathology of these forms, my observations having been mostly made on a more serious form which, for distinction, I shall call *Erysipelatoid Wound Gangrene*, using the word gangrene as if it were synonymous with necrosis.

It sometimes chanches that, in an individual whose constitution has been enfeebled by organic disease or prolonged suppuration, there occurs, around a wound accidentally received or resulting from some operation, a low septic inflammation, unmarked by much pain or heat, the foulness of the wound, the white painless cedema in its neighbourhood, the feeble pulse, the failing appetite, and the rise of temperature, chiefly calling our attention to the process that is present. The appetite and digestion are more enfeebled than impaired; the tongue, although white, is not foul; and the intellect is entirely unaffected.

Around the wound appears a redness—I had almost said a blush of redness, but it is no blush but rather brick-dust red—which extends for a considerable distance into the cedematous neighbourhood, and is marbled and mottled so that bands of pale brick-red colour embrace islands of white. The disease has no distinct edge, but presents itself in the form of patches of the size of the palm of the hand or less, which here and there, becoming livid or violet in hue, with detached epidermis and flaccid blisters containing bloody fluid, show the commencement of gangrene. That this disease is erysipelatoid in its nature cannot be doubted; the dirty wound with its gruelly discharge, the mottled red invasion of the living skin causing cedema in it and the subcutaneous tissue, spreading insidiously where life is, yet surely presaging the death of the parts it affects, mark a disease that the experienced eye finds no difficulty in recognising as related to erysipelas. It runs its stealthy course so secretly that patches of evident gangrene surprise one unexpectedly every here and there.

This disease, not uncommon in bad compound fractures, is, I believe, the most virulent and characteristic form of *poisoning*

by *streptococcus*. Although I have not had enough erysipelas material at my disposal to enable me to speak with confidence, yet I am inclined to the belief that it will be found that erysipelas and erysipelatoid diseases are all due to *streptococcus*, never to *staphylococcus*; and that this that has just been described, the most intense and fatal form of erysipelas, is a type of the whole class.

On examining the pus or grumous discharge that escapes from the wound, it is found to be extremely rich in various forms of micro-organisms, but especially to present the *streptococcus* in unusual profusion. In most wound-discharges *streptococcus* is by no means a prominent object, is indeed seldom present, but here it strikes the eye at once, chains of two to ten links lying everywhere scattered about in active growth, as their divisions testify.

In the skin and subcutaneous tissue around the wound, wherever the mottled redness exists, the microscopical appearances differ greatly from those described under the last disease. The tissues are not pervaded with the same clouds of micrococcus. Patches of tissue where cocci are proliferating occur only here and there, and have to be sought out with some care. Where they do exist they do not present the marbled bands and patches of the last disease, but fields of tissue with cells and nuclei comparatively little altered, show chains and clusters of the *chain coccus* insinuating themselves among the crevices, winding here and there, and tending little to form groups, giving to the tissues the appearance of being pervaded by fine thread-like necklaces of the micro-organism.

In the blood-vessels of the part there are occasionally to be found, generally in connection with the *tunica intima*, a few groups of the chain coccus; but as a rule the *blood-vessels* are exempt from serious participation in the disease. *This, on the contrary, centres in the lymphatic vessels* of the part. Every one of these is choked by a firm thrombus formed of fibrinous material with fibres interlacing in all directions, and containing entangled leucocytes; while the clot, that in the process of preparing the sections, has been so acted on by the reagents as to have shrunk and become detached from the vessel's wall, is pervaded with immense groups, chains, and masses of the strepto-

coccus, most abundant on its surface. Where a transverse section of a lymphatic vessel occurs it is seen that the cocci, though growing in all parts of the lymphatic clot, are chiefly on its outer layer and on the inner surface of the vessel, as if they had crept along the tunica intima of the latter, like the incrustations in a water-pipe, forming a tubular lining, and eventually obturating the vessel by means of a coagulum growing from the parietes to the centre of the vessel's lumen.

There exists, in fact, wherever the erysipelatous process occurs, a capillary lymphangitis of the part, leading to obliteration of all the lymph-passages by their being choked with coagula, and causing the death of the part by obstruction to its lymph circulation, as well as by the irritation of the poisonous ptomaines generated during the growth of the organism.

In the blood of these patients there are cocci to be found, fairly proportionate in number to the severity of the disease, and in the urine cocci, as well as rod-shaped micro-organisms, are usually readily detectable.

I have never been able to produce this disease in animals, save by the use of the discharges collected from such a case. These, however, are so virulent that there is no difficulty in producing, by injecting them under the skin, characteristic local infiltrations and general disease. Around the injection site, in patches partly cedematous, partly red and dense, partly suppurating and cheesy, the tissues are pervaded with multitudinous chains of the streptococcus, growing in the tissues and lymphatics. The blood of the animal shows a few micro-organisms, and in one case immense balls of streptococci mixed with bacilli were found among the bile in the gall-bladder. If the animal survive the first week, and, as not unfrequently happens, tend towards recovery, the blood ceases to present any micro-organisms, even though these can still be found in plenty in the infiltrated and suppurating regions around the injection site.

One feature of this disease, as it occurs in man, viz., the yellow or earthy colour of the skin, will be treated of more fully further on.

I have been guided to the conclusion that this form of disease is characteristic of poisoning by the streptococcus or chain

coccus, while that last described is typical of poisoning by the staphylococcus or grouped organism. In the former report I left unanswered the question as to whether these two organisms were different or identical. The observations that have been made since it was written have inclined me to the belief that they are separate and distinct forms. They are often found together in abscesses, and it is then impossible to gain much information regarding them, since both possess the power of causing inflammation ending in suppuration, and both cause phlegmons. But the more disease approaches the erysipelatous type and concentrates itself in the lymphatics, the more evident does its connection with the streptococcus become, while suppurative inflammation expending itself on the tissues rather than on the lymphatics seems to be the characteristic result of the staphylococcus. To put it shortly, localised phlegmon is usually due to staphylococcus, an erysipelatoid process to streptococcus. The two diseases are in many instances not very unlike to one another, but there really exists a difference between the effects of the organisms, although by no means a great or essential one.

#### SEPTICÆMIA, PYÆMIA, AND SEPTICO-PYÆMIA, AS SYMPTOMS OF MICROCOCCUS POISONING.

These are names directly suggestive of the idea that the disease for which they stand presents as one of its most marked features a condition in which the blood is adulterated or rendered impure by the presence in it of some morbid materials of a septic nature, and possessing some affinity with the unhealthy matters that exist in pus. The basis of truth that underlies this supposition has, however, gradually been brought to support a hypothesis that in the disease the entity *blood-poisoning* is fundamental and essential; that in an infective matter entering the blood and developing chiefly there, as yeast develops in a suitable liquid, is to be sought the key to a knowledge of the malady.

It has been sufficiently stated above that such a hypothesis finds no support either in a study of analogous conditions of the system or in such facts as can be made out by combined clinical and pathological observations on the disease itself.

It has been shown that the characteristic phenomena of ordinary zymotic diseases are inexplicable on the supposition of

the morbid cause having its citadel in the blood, but can be readily understood if the tissues be the entrenchments which they occupy, and the blood merely a province where a guerilla warfare is carried on.

In gradually presenting the various forms of micrococcus-poisoning in increasingly malignant phases, and noting how each presents, along with its local phenomena, the signs of a concomitant blood-poisoning that likewise rises in the scale of malignancy until it presents all the features of well-characterised septico-pyæmia, I have endeavoured to pave the way for the disease whose titles head this chapter being considered as merely part of a disease, sometimes the only part evident to our unaided senses, but in reality a subordinate part of a real disease that is somewhere raging in a local centre, could we but discover it. In this chapter I shall strive to show that not only occasionally or frequently is septico-pyæmia the companion of a local disease, but that it invariably is so, and should indeed be considered as the shadow of a disease whose substance is to be found in the tissues, without which it could have no existence.

In the ground already traversed it has been maintained that associated with all local acute suppurative inflammations there exists a feverish condition due to a slight form of septicæmia from passage of organisms and ptomaines into the blood, or in other words, that inflammatory fever is a mild form of septico-pyæmia. It has also been urged that in phlegmonous diseases, severe from the outset, or that have passed from a mild beginning into a severe after-stage, the inflammatory fever becomes more intense, presents the ordinary picture of septico-pyæmia. And the next point in the natural sequence of thought is to discuss the conditions where the disease presents so much of the general and so little of the local character as to have given rise to the assumption that the latter did not exist.

But, prior to doing so, I would wish to draw attention to a class of cases that do not belong either to what is ordinarily understood as acute suppurative inflammations on the one hand, or septico-pyæmia on the other, and which yet throw much light on both. The disease called *malignant lip pustule*, generally looked upon as a malady *sui generis*, is an instance of a highly fatal disease where a local focus presenting at first the pheno-

mena of inflammation hurries rapidly on to the stage of suppurative infiltration or gangrene, while coincidently there occurs a general condition of blood-poisoning that tends to prove rapidly fatal. The local centre of disease, like an ordinary acute inflammation, is at first taken for nothing more, but combines a rapid unfolding of local virulence, evidenced by its quick extension into the healthy neighbourhood, and the rapidity of its passage through the ordinary stages of inflammation to terminate in slough or suppuration, with the development of general symptoms indicative of a serious poisoning of the system.

It seems as if some toxic agency akin to that of typhus and typhoid fevers were at work, and rapidly led to the production of a *typhoid* state, where high fever and disorders of the nerve centres created the most imminent danger to the life of the individual. Malignant lip pustule occupies a highly vascular site, whence rapid penetration into the system is greatly favoured. It indeed has all the outward character of a *phlegmon*, with in addition grave general symptoms, and its names all point to this resemblance (carbuncle anthrax). Nor is this to be wondered at, for it is in all probability nothing more than a virulent phlegmon in a vascular region, and occurring in a subject of low vitality and diminished resistancy. In the one case, where I had occasion to examine the disease, it occurred in a male aged forty-five, a patient of Dr. Trail of Strichen. It consisted of a spongy purulent infiltration of the whole thickness and length of the lower lip for a full finger's breadth from the margin downwards. The general symptoms were most alarming, the patient's life was in serious danger when I saw him, on the eighth day of the disease. The lip was pervaded with innumerable sloughs and miliary abscesses, and these crowned with groups of staphylococci, each coccus averaging  $\frac{1}{480}$  of a millimetre in diameter. There were no streptococci present. The pus was estimated to contain 90,000 cocci in each cubic millimetre. One half minim injected under the dorsal skin of a large mouse killed it in forty-eight hours, with the usual symptoms of septicæmia, and the cedematous injection site showed one gigantic colony of grouped coccus not yet reaching the stage of suppuration, while the blood contained similar cocci in less abundance. The blood from the heart was unable to

produce any infection when rubbed into shallow scratches on the ear of a stout guinea-pig. The patient eventually recovered by thoroughly drenching the tissues of the lip with carbolic acid solution slowly injected into it through numerous punctures while he was under the influence of chloroform. In my judgment, this man's disease was a very virulent staphylococcus colonisation in a vascular region, where, either owing to defective resisting powers or to extreme virulence of the micro-organism, there was little attempt at limitation of the process by granulation tissue being formed around it, and hence the system was poisoned, more however by the ptomaines passing into it than by the individual cocci that strayed into the blood. These latter seemed to have found the blood unsuitable for their existence, as was shown by the absence of any secondary foci in any other part.

*Acute infectious osteomyelitis* is another disease where septicæmia exists along with a well-marked local centre whence it springs. Of this disease I have seen but one example of late, and it occurred in the practice of Dr. Williams of Tarland. A young boy, naturally delicate, and who had been exposing himself for hours to the intense cold of the winter of 1880-81, was taken with rigors and pains in his right tibia and left ribs, rapidly developing into a well-marked acute inflammation of the medulla of the tibia. The symptoms over the tibia were heat, slight redness, great swelling, and intense pain; the ribs presented a similar affection complicated with pleuro-pneumonia, and the general symptoms were of the severest typhoid type, with high fever, ending in stupor and death. When an incision was made over the lower part of the tibia, where the greatest pain was complained of, and a drill sent through the compact layers of the bone, the usual oily pus of osteomyelitis squirted out, showing the great pressure it had been subjected to, and was found to contain large numbers of the staphylococcus. The evacuation of the pus was not followed by much improvement in the general symptoms, although it relieved the local pain. It was unfortunately not in my power to obtain any of this pus for an experimental investigation. The facts of the case suggested the following explanation. This boy, constitutionally feeble, chilled by long periods of exposure to the intense frost, had the



forces of his tissues so weakened that invasion of micro-organisms from without or from the intestinal tract had occurred in his system. Of these organisms the staphylococcus, developing in his weakened frame, had found a suitable resting-place in the medulla of his ribs and tibia. There it had developed into colonies, that produced their usual effect, viz., acute inflammation, and from thence the ptomaine-poisoning had occurred that eventually caused his death.

These two diseases will serve as types where we can see septicæmia and pyæmia, in undeniable clearness and purity, yet dependent upon a local disease that cannot be explained away as being other than their true cause and starting-point, and will illustrate the questions that confront us in dealing with septicopyæmia.

There is no such disease as septicæmia or pyæmia *per se*, such conditions are merely secondary in the order of the morbid process and depend on the existence of local foci of micrococcus growth. They are but the expressions of malign influences coming from this focus, and would in every case cease to exist were it in our power to remove or cure the focus. From textbooks and monographs, not from nature, we have learned to believe in them as possessing an individual existence. But is this correct? There is no observer, accustomed to observe with care and ponder with earnestness, and whose pathological knowledge has kept pace with his clinical study, who will be able to assert that he has seen a case of septicæmia existing alone. I have never personally met with such. In the work of the surgical wards, as in the more varied experiences of general practice, it has always been the reverse: individuals who have been injured or operated on, or who have become the subjects of acute inflammation or gangrene, or who have presented some lesion or wound whence the process had originated, have been the only ones who have exhibited septicæmia or pyæmia. And wherever there has appeared an acutely inflammatory disease, whether arising by seeming spontaneity or beginning in a wound, there, so surely as the signs of general invasion were present and the thermometer in the axilla showed a temperature considerably exceeding 101° Fahr., did the microscope reveal in the local discharge the existence of micrococcus, and its presence

to a less extent in the blood. If the patient grew worse, then the local discharges showed a greater abundance of the cocci, and the blood became more pervaded with them, the intensity of the local inflammation and of the general signs corresponding with the numbers of these organisms present, and the signs of their active growth as shown in their rapid division. Did the patient tend towards recovery, the fact was signalised by a diminution in their number, as well as by the favourable conditions of pulse, temperature, and functions that are familiar to us all.

It will occasionally happen that the general symptoms are very marked, while the local disease may be hard to discover. Not every large colony of micrococcus signalises its presence by the clamant signs of redness, heat, pain, and swelling; on the contrary, where the tissues or the individual affected are weak, the growth and invasion may proceed with such astounding celerity that large tracts are invaded and the system is saturated with the ptomaines ere we are well aware that anything is going wrong. Even then, care will detect the local site of infection. Experiment on animals shows that if micrococci that have been cultivated into great virulence, such as those taken from a virulent suppuration, or, as stated above, from a malignant phlegmon, be injected, especially into a weakly animal, it exhibits a disease that looks like septicæmia; it fevers, its nerve centres become intoxicated, it is dull and listless, and in a few hours it dies; everything pointing to the ailment of which it perishes being generalised in its system, while at the injection site a little puffiness may be all we discover. Yet when the animal is subjected to microscopic scrutiny, we find in the seemingly unimportant injection site an enormous and appalling growth of the deadly organism, in quantities that defy calculation, in numbers that would be but faintly computed by millions, while the blood which we would naturally have supposed to be richly inhabited, is so poorly provided with the organisms that it is clear that the chemical intoxication by the ptomaine, not the vital injection by the germ, has been the cause of death. The local invasion has been so swift that the symptoms betraying it have been but few. In my former report I have mentioned the case of a man where septicæmia followed

an extirpation of goitre, as well as a case of compound dislocation and fracture where the microscopic investigation was full and convincing, and I have other cases since that report was written that tell the same story.

Where cases of septicæmia end, not in death but in recovery, the part played by the local focus usually becomes evident, for suppuration and inflammation, it may be gangrene and necrosis, arise in and around it, so that the poisonous centre that may once have presented so few symptoms is made plain, and health is not regained until it is extruded by the agencies of reaction and repair.

It cannot be needful that all the gradations between the most intense forms of disease where the general symptoms are striking and the local ones obscure, and the slightest forms where the local symptoms are patent and the general almost or altogether undistinguishable, be again commented upon in detail. The matters previously gone over are sufficiently illustrative, and I may now simply refer to these as warranting us in concluding that phlegmon and septicæmia are but one disease, produced by micrococcus poisoning in some of its varying degrees of intensity. To use the language of Kocher, "between a simple localised acute inflammation on the one hand, and the severest case of pyæmia, there exists only a difference in degree, a difference in intensity."<sup>1</sup>

Phlegmonous inflammation, septicæmia, pyæmia, and septico-pyæmia are all micrococcus poisoning, varied, however, according as ptomaine intoxication or the local tissue reaction becomes more prominent. Every feverishness, from an inflamed throat or finger, is a septicæmia in a mild degree, and may pass into a severe form. Ptomaines pass into the blood, and coincidentally a few individuals of the micrococcus may be found to have wandered from the local disease and to be circulating in the blood, dead or half-dead, owing to the unsuitability of the medium where they are and the unfavourable influences of the forces of the tissues. If removed from the blood they rarely grow when put into a suitable medium. They are all eventually extruded or consumed. But if the individual be subjected to

<sup>1</sup> Kocher, "Aetiologie der Acuten Entzündungen," Langenbeck's *Archiv*, vol. xxiii. p. 103, 1879.

depressing influences, the ptomaine poisoning may not be the only phenomenon observed. As the symptoms become more severe and the micrococci more numerous in the blood, the weakness of the individual becomes greater and the resisting power of his tissues less, so that the micrococci are able to live in the blood, where previously they found this impossible. They multiply and form small groups that increase in size until they are too large to pass through the capillary network, and therefore are caught and detained in lungs, liver, or some other part. There they continue to increase during life, perhaps even for a time after death, and furnish their contribution of poison to the system.

Or it may be that, though unable to multiply in the blood, they here and there throughout the body find spots suitable for their development, where they can multiply and form the foci of suppuration that mark the form for which we usually reserve the name *pyæmia*. The pyæmic secondary foci are usually in lung or liver, or joint, but may equally well occur in lymphatic glands, secreting glands, or even in connective tissues. They are but a rehearsal of the local growth and infiltration already described in the primary focus, and require no separate description. In the joint affections of pyæmia, however, there has seemed to me to be something peculiar. It would naturally be imagined that the organisms in the blood, finding their way into the quiet haven afforded by the joint cavity with its tranquil pool of synovia, would multiply in that liquid until they and their ptomaines were sufficiently numerous and intense to provoke reaction, when the phenomena of arthritis might be expected to appear. But this does not seem to be their course. In one pyæmic case, when I drew off by the aspirator a little serum from a knee-joint at an early stage of the disease, there were no micro-organisms in the fluid, and it was only at a later period, when it was becoming purulent in character, that these appeared. From this I surmised, perhaps incorrectly, that pyæmic arthritis is not due to organisms multiplying in the cavity, but somewhere in its neighbourhood, possibly in the delicate synovial fringes around the cartilages where the capillary network is very fine, and that the early liquid in the joint is merely the serum or synovia effused from the outer surface of

the spot where the colony of micrococcus is still in a comparatively unadvanced stage of growth.

There is one phenomenon common both to septicæmia and pyæmia that requires to be mentioned here, although I regret that my observations concerning it are so barren of result. I refer to the *yellow colour* of the skin observed in severe cases. This symptom has been merely mentioned in the portions of this report devoted to sloughing inflammation and erysipelatoid disease. But it is common to all severe forms of micrococcus disease. It shows itself as a paleness or earthy tint of the skin, nails, gums, and conjunctiva, that may vary in depth of colour up to a distinctly icteric tint. It differs from ordinary jaundice, however, in not affecting the urine. There is plainly some connection between this colour and micrococcus. In wounds that contain micro-organisms, and that are covered by Lister's dressings, it is very common to remark portions of the gauze tinted of a rich yellow or orange hue, and I was early led to observe that a microscopic examination of these patches never failed to reveal the presence of micrococcus. So invariable was their connection that it was evident the colour depended on the presence of micrococci. It occasionally chanced that it was observable on other dressings, or even in the pus of a wound, but wherever it was seen I have always detected large quantities of micrococci. I cannot say that the wounds where this orange colour occurred have given evidence of being more dangerous than other wounds, although it were well not to forget the statement of Verneuil,<sup>1</sup> that such wounds are apt to be associated with dangerous forms of blood-poisoning. The orange colour indicates the presence of micrococci, and therefore such wounds are not pure; danger lurks in them, but the regular use of antiseptics and drainage removes the danger. In cultivating micrococci the yellow tinge was sometimes obtained. This was rarely the case, and the colour was but faint in albuminous fluids exposed to the air, but in the interior of eggs it was sometimes very marked. It was not much seen when the streptococcus that rapidly and uniformly pervades the whole egg was employed; indeed the yellow tinge then seen in the albumen may have been due to the admixture of the yolk that results from the agitation of the all-pervading growth.

<sup>1</sup> *Archives gén. de. Méd.*, December 1880, p. 641, "De la Suppuration orangée."

But where the staphylococcus was employed a different style of growth and a very marked coloration was several times observed. Staphylococcus grows at the injection site, not tending to spread rapidly, but rather to mat all that lies around it into a firm turf or clod, and the resulting clot-like turf was in several instances found of the deepest orange yellow next the shell, as if the colour were the result of the growth of the organism under peculiar conditions. Hence it appears possible that the yellow or earthy tinge of the surface observable in cases of septicæmia may be in part at least due to the production of a yellow pigment by the micrococcus.

Should the views here advanced that septicopyæmia is simply micrococcus poisoning prove to be correct, it will be necessary to go a step farther and admit that *hectic fever*, that septicæmic condition that differs from ordinary septicæmia in being associated with *ulcerative phthisical* processes in the lungs, and with ulcerative diseases in general, rather than with wounds and operative procedures, is but a form of the same disease. This I am prepared to assert. Although in the hectic of phthisis we have to deal with a milder and more protracted, although equally fatal form, yet there is no clinical difference in the appearances or symptoms from those that occur in septicæmia from advanced disease of the hip-joint or spine, or in the suppuration resulting from a bad compound fracture not treated antiseptically. The two diseases are absolutely identical, and surgical septicæmia and hectic fever are really synonymous terms. Hectic fever only occurs where there is a tendency to suppuration; it does not exist in phthisis until suppurative breaking down of the lung occurs, and micrococci are always present in the expectoration of such cases. Without septic suppuration, hectic fever never exists. In tubercular maladies there is no reason to suppose that the micrococci are other than ingrafted on the diseases when they have reached a certain stage, having been conveyed into the lungs by inspiration, or through the circulation, and similarly appearing in chronic suppurations about pelvis, spine, or joints, either by the rare penetration into them from within or the more common introduction from without. Finding in the weakened tissues a spot where their development is more or less favoured, they secure at first perhaps a scanty foothold, and commence only,

when the prostration of the individual permits their considerable extension, to show the more serious symptoms of their presence.

One of the chief difficulties in the way of admitting what is here claimed as due to micrococcus will, in all probability, be that it is not readily conceivable how one organism should produce such variety of disease. Yet a little consideration will make it plain that all the variations in the nature and symptoms of the diseases produced may be due to—1st, the difference in the form of the organism; 2nd, differences in the organ or structures invaded; 3rd, the difference in the virulence of the organism; and 4th, the different susceptibility of the individuals attacked.

1. *On the difference in the form of the organism.*—I have already dwelt on the modes of growth of the streptococcus and staphylococcus, and their correspondence with certain varieties of disease. It may be true or not that time will reveal further differences among the micrococci found in the pathological conditions; this I cannot tell. It may also well be true that there are other micro-organisms besides micrococcus that can give rise to acute inflammations and septicopyæmic conditions; this also I cannot tell; I only know that they must be rare. Last year Professor Ewart put on record an investigation into an epidemic of sore throat, fever, and rheumatic pains, followed in a few instances by local suppurations, seemingly due to a rod-shaped bacillus, not a spherical coccus.<sup>1</sup> Febrile maladies, however, often open a door for the entrance of micrococcus, so that abscesses and inflammations due to the latter are common as sequelæ of the former, and this renders the question a hard one to decide. It is certain that septicæmia in animals is not usually due to micrococcus, but to other organisms; and these may be capable of occasionally appearing in man. But, with these reservations, I do not hesitate to say that micrococcus is the cause of all septicopyæmia, as of all phlegmonous inflammations in man.

2. *The differences in the organ or structure affected* may well be expected to explain to a considerable extent the clinical ap-

<sup>1</sup> *Proceedings of Royal Society of London*, 1881, No. 215. A new form of febrile disease associated with an organism distributed with milk.

pearances seen in the diseases due to micrococci. Inflammation of a lung, a serous cavity, an eyeball, or a vein, cannot be expected to give rise to identical symptoms, and such considerations as belong to locality will explain much that would otherwise be startling in the extensive range of diseases caused by these organisms.

3. *The varying virulence of the organism* seems to play a large part in the multitude of disease-forms it originates. Micrococcus, as it exists on the surfaces of our bodies and in decomposing fluids, is a comparatively innocent germ, that may, in the state and quantity it there exhibits, be injected under our skin with impunity. I have never succeeded, I freely admit, in cultivating such cocci into the virulence sufficient to produce inflammation and disease, although I have often reduced virulent cocci into a harmless condition by growing them with free access of air. Yet, despite my failures, I believe that they are one, and that it will yet be found possible, by growing them under proper conditions, to demonstrate their identity. From the experiments mentioned in my former report, it seems probable that cultivation in animal fluids and exclusion of air are the chief conditions requisite to bring forth their virulence; while this is diminished by being grown in fluids not of an animal nature, and under free access of air. All that has been observed concerning them, experiments made with them, and their behaviour as seen in disease, seem to favour this view.

If analogy may be claimed as giving strength to such a supposition, it points entirely towards culture evolving virulence. It is now a well-worn observation that harmless organisms can be made deadly, and deadly ones harmless. Grawitz cultivated innocent fungi into virulence; and in Buchner's experiments we can see the common hay-bacillus growing into a troublesome, an alarming, a most dangerous, and lastly a certainly fatal organism, more lethal than the most subtle chemical poison. In the great facts of protective vaccine that we owe to Pasteur and others, we find the converse process laid bare to us, and can observe how, with the diminishing virulence from suitable culture, the properties of an organism become marvellously changed. Pasteur says, concerning the organism of splenic fever<sup>1</sup>—"The small

<sup>1</sup> *Comptes rendus du Congrès International des stations agronomiques*, Paris, 1881, pp. 154, 155.



filamentous organism of anthrax readily produces spores. It generally grows by dividing, like the little organism of fowl-cholera; but after twenty-four or forty-eight hours, especially if grown with abundant exposure to air, which it requires if it is to grow by fission, there appear in the slender filaments composing it small brilliant points, its spores; around these the filament becomes absorbed and soon disappears, leaving only a cloud of minute brilliant granules. This is but another mode of generation of the anthrax parasite; for if you take these brilliant grains and place them where they can grow, they immediately reproduce the filamentous organism that grows anew by fission for several days, then again forms spores, and so on. But here is a strange peculiarity: when the spore forms in the rod it possesses the exact virulence of the rod or filament; that is to say, if you cultivate the blood of an animal that has died spontaneously of anthrax till in twenty-four or forty-eight hours it forms spores, you will find, if you test the virulence of these germs, that it is identical with that of the blood from which they were produced.

"There are two circumstances under which the small filamentous organism can develop without producing spores,—a very low temperature, about 60° Fahr., and a very high one, about 110° Fahr.

"Bring the liquid where the anthrax is sown to a temperature of 104°, or perhaps 20° or 30° below that point, and it grows and forms spores; but at the temperature of 110° Fahr. it grows quite as well as at the lower temperature, only it forms no spores.

"Now leave it at this temperature, so that it is exposed at the same time to the action of the oxygen in the atmosphere of the vessel, and then do as you did in studying the organism of fowl-cholera, that is, studying the effect of time on its virulence. When you have kept the flask at 110° Fahr. for four, five, or six days, you test its virulence, and find that it already gives evident signs of diminution; in eight days this is more manifest, and in ten days, fifteen days, or a month, it becomes step by step more feeble. After a certain lapse of time it is all dead, the organism will not respond to further culture; but before it dies, if you test its virulence, you will find you can kill neither guinea-pigs,

sheep, nor rabbits; and in the interval of time that has elapsed between the exposure to the temperature of 110° Fahr. and this extreme period of a month or six weeks, you have *as many viruses differing in their virulence as you have days.*"<sup>1</sup>

In the face of these statements concerning one organism, it is not an extraordinary thing to assume that the same may be true of another, and that the micrococcus may vary greatly in its virulence.

4. But *the different susceptibility of the individual* probably plays the most important part in varying the forms and intensity of micrococcus poisoning. If the same dose of micrococcus pus be injected into each of a number of mice of the same litter, the effects of the dose will greatly vary—perhaps one, the largest and strongest, may escape unscathed or be but slightly ill, in several others abscesses will follow, in some necrosis, and in one perhaps, the smallest or most weakly, death from septicæmia results. Here the evidence of individual susceptibility is very strong.

When we reflect on what we know of other diseases, it becomes even stronger. In an epidemic of measles or scarlatina all are not affected alike; some are seriously ill and die, some are trivially affected, others entirely escape and will not take the disease. A family of children, constitutionally strong, takes the disease and considers it a pastime; a hundred yards off, a delicate family also takes it, and several of its members die; while a third family, naturally healthy, but living in a badly-drained house, also pays a toll of several lives.

And not only does intensity seem to be modified by constitution, but the very form of the disease can be altered by it. In typhoid fever an individual will have an attack of the disease lasting three weeks, another six weeks, a third will have no diarrhoea, a fourth will simulate acute bronchitis or acute disseminated tuberculosis, a fifth will be struck down comatose, as if from apoplexy, at the very commencement, and so on.

<sup>1</sup> M. Pasteur goes on to show how each of these degrees of virulence can be definitely fixed by transferring the organism to a temperature in which spores are formed, when these at once appear, possess exactly the virulence the bacilli had got to, and keep it indefinitely, so that a known degree of virulence can be sent to the ends of the earth.

Hæmorrhagic smallpox differs from common variola only in the individual constitution of its victim, and yet how unlike are the diseases. It were easy to multiply evidence of the great part played by individual constitution in modifying the forms assumed by the same bacteric diseases.

The agencies at work in modifying the intensity, situation, or extent of micrococcus poisoning are so potent, that there will be found to be nothing in the variety of its forms and stages that does not readily harmonise with the assumption of their being due to but one disease.

It is greatly to be regretted that additions to our knowledge do not immediately result in additions to our means of treatment. We are still far from possessing a cure for septic diseases. Yet there is some gain in the way of treatment to be derived from these views, should they prove to be true. We must, in the first place, abandon the idea that by the internal administration of general remedies we can eradicate from the blood or neutralise in it diseases whose essence is not to be found there, but elsewhere. We must zealously fall back on our local measures, striving to prevent the introduction of germs, and to destroy the local colonies of those that have entered. The present is an epoch when it seems somewhat unfashionable to be a thorough antiseptician in theory and practice, when such words as bacteria and carbolic acid have become commonplace and vulgar, and when there is danger of reaction carrying us back towards our old aimlessness in treatment.

Human nature forgets unseen foes, but were every surgeon and physician familiar with the microscopic study of micro-organisms, then, dealing as we would with visible realities, and beholding both our faults and their punishment, in the treatment of wounds and disease, it would be less easy for fashion to mislead or prejudice to warp our minds.

## OMPHALO-MESENTERIC REMAINS IN MAMMALS.

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WHILST making investigations concerning the peritoneal arrangements in some of the mammalia, I have noted in new-born Carnivora that, in addition to the obliterated cords of the hypogastric arteries and umbilical vein, two other more slender cords exist in the abdomen. The additional cords do not, however, run outside the peritoneal sac, but pass directly through its cavity, hence arises a difficulty in preserving them, when, in order to see the connections of the cords, the intestines are moved about.

The cords are attached below to the abdominal surface of the umbilicus, and from this they pass forwards and upwards, keeping close together at first, but afterwards separating. The upper end of one passes into the mesentery of the *ilium* close to its intestinal border, the other passes up to the *mesoduodenum*, and runs into it, close to one of the pancreatic lobes.

On dissection, the posterior cord is found to be connected with the mesenteric artery, the anterior one with the portal vein. I have found the cords in every specimen of new-born cat and dog examined, and frequently in these animals when a week or a fortnight old, and in a few cases at even a greater age. I have also seen them in a young lion which lived for seven weeks, but, judging from the domestic Carnivora, such a long persistence of the cords is exceptional; it seems very likely, therefore, that in this case the cords were retained simply from feebleness of the animal associated with his birth in a state of captivity. The disappearance of the cords is no doubt brought about by their rupture, after which they become absorbed.

These peculiar abdominal cords (existing *post-partum*) are not, however, characteristic of the class Carnivora, for they are present also in new-born animals of another group. They can be very distinctly seen in the guinea-pig up to a week old, probably longer, and here also their connections and course are the same as in the kitten, &c. In the young rabbit there is a somewhat similar arrangement, but in it I have not, up to the present, suc-

ceeded in finding after birth more than one cord, and it, like the anterior of the two cords in the guinea-pig, is traceable up to the portal vein.<sup>1</sup>

That the cords are remains of the *omphalo-mesenteric* system of vessels is shown by their connections, the one which is attached to the *mesenteric* artery being the obliterated *omphalo-mesenteric* artery, the cord which goes to the *portal* vein being the remains of the *omphalo-mesenteric* vein. The correctness of this interpretation is proved by the dissection *in situ* of *intra-uterine* specimens of any of these animals, *e.g.*, the foetal guinea-pig, the cords can then be traced out of the body of the foetus, through the umbilicus, and into the umbilical vesicle.

It is at first difficult to conceive how it is that, in a mammal where the use of the yolk sac is so evanescent and soon dispensed with, the vessels, whose sole function is to absorb from the yolk, are retained for such a great length of time as to present themselves after birth as cords of considerable firmness. In looking for an explanation one is apt to have recourse to a study of the phases of development in a *mesoblastic* ovum, but, in the chick at the time of maturity, and for some days prior to that, the only vessels which pass to the umbilicus are those connected with the allantoic circulation. Neither the pedicel of the yolk sac nor its vessels take part in the umbilical cicatrix in the bird, inasmuch as for some days prior to the completion of the hatching process the remains of the yolk and its vessels were drawn up into the abdominal cavity of the chick, the absorbing process continuing uninterruptedly. This "sucking up" of the yolk was not interfered with by the allantois, there being no vascular or other adhesion between them. On the other hand, in mammals the yolk sac is not drawn into the abdomen, and in some cases the envelope of the yolk continues to grow after its nutritious contents are absorbed.<sup>2</sup> What I

<sup>1</sup> I have not found them in the new-born rat; they are therefore not present in all rodents after birth.

<sup>2</sup> The persistence of the sac of the umbilical vesicle is characteristic of the Carnivora, Insectivora, and Rodentia, and a knowledge of this circumstance has led Von Beer, in 1828, to divide the mammalia into two great groups, viz., (1) those in whom the vesicle persists, and (2) those in whom it soon disappears.—*Ueber Entwicklungsgeschichte der Thiere*, Erster Theile, p. 225. Also see drawing from Buffon and Daubenton, in Mivart's *The Cat*, p. 328.

would therefore venture to suggest, as explanatory of this, is that an adhesion had formed between the allantois and yolk sac, and that when this adhesion was great an intercommunication was formed between the vessels of the two sacs, and that this led to the retention of the *omphalo-mesenteric* trunks long after the cessation of their primitive function. Where both cords are retained (kitten, puppy, lion, cub, and young guinea-pig), there has been a communication between both the arteries and veins of the two systems, but where only a single cord exists after birth (young rabbit) the veins only of the two sacs have been connected together.

It is a curious fact that, while the vessels of the yolk sac are, in those cases, preserved for such a great length of time, the vitelline duct has disappeared before birth. This, which at first sight is inexplicable, is probably dependent on that which led to the retention of the vessels, and may be explained in this way: the umbilical intestinal loop is drawn into the abdominal cavity as the rest of the alimentary canal grows; this movement, then, on account of the fixed position of the umbilical vesicle outside, causes the vitelline duct to be stretched, so that it very probably soon ruptures, and is then speedily absorbed.

In the higher mammals the cords exist only in very early foetal life. I have been able to trace them in a human foetus which measured three inches in length from vertex to coccyx, but they were of an extreme degree of tenuity, and did not admit of dissection up to their pervious ends.

THE ACTION OF SALINE CATHARTICS. By MATTHEW HAY, M.D., *Demonstrator of Practical Materia Medica in the University of Edinburgh.*

(Continued from vol. xvi. p. 604.)

SERIES OF EXPERIMENTS, F.

On the purgative effect of the salt when injected subcutaneously.

After the tolerably exhaustive inquiry which I have made into the action of the salt when directly introduced into the circulation, it may appear superfluous to investigate the effect of the salt when injected subcutaneously. For whether a soluble salt be injected into a vein or beneath the skin, the belief is that it produces its peculiar action, if not equally quickly, at least as equally powerfully, the salt passing rapidly, in the latter case, from the subcutaneous tissue into the circulation. It was, therefore, to be expected that the action of the salt would be the same in both cases. And so it was believed, until Luton, an eminent physician of Reims, accidentally observed that one decigramme ( $1\frac{1}{2}$  grains) of sulphate of magnesia could produce purgation when injected subcutaneously. I have not had an opportunity of consulting the *Bulletin de la Soc. Méd. de Reims*, in which his communication appeared, but have seen, what I take to be, a fairly complete résumé of it in the *Gazette hebdomadaire* (1874, p. 455). Four experiments were altogether made, and all of them on man, and in each instance free purgation followed. Unfortunately, the situation of the injection is not mentioned, but I presume it was made beneath the skin of the abdomen, the situation chosen by Vulpian and Carville,<sup>1</sup> when, a few months later, they repeated with the same result these experiments on dogs. The original communication of Luton is, as the editor of the *Gazette hebdomadaire* remarks, silent as to the production of pain at the seat of injection. In Vulpian and Carville's experiments a considerable degree of pain and inflammatory swelling followed; and it

<sup>1</sup> Vulpian, *Leçons sur l'appareil vasomoteur*, t. i. p. 515; Carville, *Gaz. hebdom.*, 1874, p. 405 *et supra*, p. 8.

is not improbable that this was also observed by Luton, although not recorded. In attempting to explain the powerful action of so remarkably small a dose of sulphate of magnesia, Vulpian believes that the salt is absorbed and effects catharsis by its action on some portion of the intestinal or alimentary mechanism. But it is passing strange that, when the same salt is injected into the blood, purgation does not follow. And Vulpian, as he himself admits, injected directly into the circulation both large and moderate doses of sulphate of soda without producing any effect. In spite of these considerations Vulpian, in common with Luton and Carville, adheres to the view he originally stated.

The activity ascribed to the absorption of an almost infinitesimal dose of sulphate of magnesia seemed so incredible that Gubler attributed the purgative effect in the case of man, not to the injection of the salt, but to the mere anticipation of such a result, the individual having been informed that purgation was expected. This, in the less scientific and cultivated minds of the inmates of an hospital, where as yet all the experiments on man had been made, Gubler considered very likely to occur. To test the truth of his supposition, he administered the salt in the same manner to a large number of the students attending his clinique, upon whom the moral effect of the injection was reduced to a minimum. The result was highly favourable to his theory. For only in a very small percentage of those submitted to the action of the salt did purgation occur. The seat of the injection appears to have been the arm. The dogs could not have been led to expect purgation from a subcutaneous injection, and Vulpian and Carville never failed to observe purgation in all the dogs with which they experimented. Moreover, the latter, as I have mentioned in an earlier part of this paper,<sup>1</sup> found, when he killed the animals before purgation had occurred, that the mucous membrane of the whole intestine was congested and inflamed, and covered with a sanguineous effusion. Evidently, therefore, Gubler's theory was insufficient.

It occurred to me that the true explanation of the action of a saline cathartic, when injected subcutaneously, lay in the salt producing irritation at the seat of its injection, which irritation through a form of reflex mechanism excited the peristaltic or

<sup>1</sup> *Supra*, p. 250.



secretory activity of the intestinal canal. The principle of treatment by counter irritation must be founded on the recognition of an intimate nervous relation or sympathy between certain internal parts and other external, generally superjacent, parts. Between the parietes of the abdomen and its viscera such a nervous relation probably exists. It is this relation which is disturbed by the irritation of the injected salt. If the salt be injected in any other situation than beneath some portion of the skin in nervous relation with the abdominal contents, and that is probably any portion of the integument other than that which covers the abdomen, then purgation will not follow, for the nervous mechanism of the intestines is not disturbed. Hence, although Luton, Vulpian, and Carville invariably observed purgation occur when the salt was injected over the abdomen, yet Gubler's experiments were negative when the seat of the injection was the arm. This is more clearly proved by my own experiments, in which the situation of the injection was varied, and in which the following points will be proved:—(1) A purgative salt is inactive when it is injected beneath the skin of the arm or leg; (2) when injected over the abdomen its cathartic effect depends upon its producing a certain degree of local irritation and inflammation, for when evidence of irritation is absent there is no purgation; (3) non-purgative salts, and one which, as chloride of sodium, exists naturally in large quantity in the blood, or one, as sulphate of zinc, which is astringent, also produce purgation, if their injection has been followed by local irritation.

*Experiment LXXIX.*—Terrier bitch, weighing 5·66 kilogrammes. Usual diet of bread, boiled flesh, and water continued throughout this and following experiments. Injected, in the forenoon, beneath the skin of the *back* between the scapulæ 0·2 gramme of crystalline *sulphate of soda*, dissolved in 2 c.c. of water.

During the following night a small quantity of hard fæces was evacuated. On the second night a similar evacuation again occurred.

There was not, at any time, perceptible swelling or inflammation of the subcutaneous tissue at the seat of the injection.

*Experiment LXXX.*—Same dog. Injected beneath the skin of the *abdomen*, two inches to the left side of the linea alba and one inch beneath the umbilicus, 0·1 gramme of *sulphate of soda* dissolved in 2 c.c. of water.

Neither during the following night or day did the animal defæcate. On the following night there was a stool of ordinary firm consistence.

There was no evidence of inflammation or pain at the seat of the injection.

Apparently in this dog sulphate of soda was incapable of purging, whether injected beneath the skin of the back or of the abdomen. It is, however, worthy of remark that in neither case was there any local irritation produced by the injection.

*Experiment LXXXI.*—Same dog: several days after previous experiment. Injected subcutaneously over the *abdomen*, inside the left flank, 0·1 gramme of *chloride of sodium*, dissolved in 2 c.c. of water.

The first evacuation occurred on the third day, and was of normal consistence.

There was no visible trace of inflammation at the seat of injection.

*Experiment LXXXII.*—Same dog. Injected subcutaneously over the outer side of the upper part of the left *fore-leg*, 2·4 c.c. of a solution of *sulphate of magnesia* containing 0·12 gramme of the salt.

There was a stool next morning, but small in quantity and perfectly firm. There was no further evacuation during the day.

There was no swelling at the seat of injection.

*Experiment LXXXIII.*—Same dog. Injected a similar quantity of the same solution of *sulphate of magnesia*, as used in the previous experiment, over the *abdomen*, inside the left flank.

Not until the second night was there any evacuation, and it was then small in quantity and quite hard in consistence.

There was no palpable or visible swelling or inflammation at the seat of injection, and apparently no pain.

*Experiment LXXXIV.*—Same dog. Injected, a few days afterwards, in the same situation, a like quantity of a solution of *sulphate of magnesia*, but double its strength, and containing, therefore, 0·24 gramme of the salt.

Although kept under observation for two days afterwards, no discharge of *fæces* took place during that time.

There was no detectable swelling of the injected part.

The sulphate of magnesia, therefore, like the sulphate of soda, failed to produce purgation, or even the slightest laxative effect, when injected subcutaneously in the same quantity and strength of solution as was used by Vulpian, not even when injected over the abdomen. Here, also, as with sulphate of soda, the injection was unaccompanied by local inflammation; and I have no doubt that this accounts for the negative results of my experiments as compared with those of Vulpian.

If these purgative salts failed to act, it was hardly to be

expected that chloride of sodium could do otherwise, as the single experiment with it shows.

A few experiments were now made on cats.

*Experiment LXXXV.*—Cat, male, weighing 2·80 kilogrammes. Injected over the *back*, between the scapulæ, 0·15 gramme of *sulphate of soda*, dissolved in 1·5 c.c. of water.

No evacuation occurred during the two following days. When it did occur the discharge was hard.

No swelling or redness at point of injection.

*Experiment LXXXVI.*—Same cat. Injected beneath the skin of the *abdomen* 0·15 gramme of *sulphate of soda*, dissolved in 1·5 c.c. of water.

During the immediately succeeding night there was a fairly large and firm stool, followed by another firm evacuation on the next night.

No signs of irritation at the seat of injection.

The result of these two experiments with this cat and sulphate of soda is the same as of those with the dog—entirely negative.

*Experiment LXXXVII.*—Cat, female; weighing 1·64 kilogrammes. Passed a quantity of perfectly firm fæces immediately before the commencement of the experiment. At 1.10 P.M. injected subcutaneously over *abdomen*, near the umbilicus, 0·15 gramme of *chloride of sodium*, dissolved in 1·5 c.c. of water.

During the night a soft pultaceous stool was evacuated of the usual colour. On the following night there was another somewhat soft stool, but firmer than the other. By the next day the fæces had resumed their usual consistence.

A slight thickening of the subcutaneous tissue could be felt at the seat of injection, and was evidently painful on pressure.

*Experiment LXXXVIII.*—Same cat; a few days afterwards. Injected over the *back*, between the shoulders, 0·15 gramme of *sulphate of soda*, dissolved in 1·5 c.c. of water.

The first stool was evacuated on the second night after the injection, and was of perfectly firm consistence.

There was some swelling and pain in the neighbourhood of the injection.

These last two experiments were more successful than the others, inasmuch as in the one, by the abdominal subcutaneous injection of chloride of sodium, a purgative effect was obtained, and in both a certain degree of inflammatory irritation was produced at the seat of the injection. This local irritation, and not the chloride of sodium, was evidently the cause of the purgation in the one experiment. In the other, where the injection was

made over the back, although sulphate of soda, a more purgative salt, was used, and notwithstanding that a similar degree of local irritation was induced, there was not the slightest laxative effect. These results certainly favour the theory I have advanced, as also do the following experiments with another cat.

*Experiment LXXXIX.*—Cat, male, weighing 3.05 kilogrammes. At 1.50 p.m. injected beneath the skin of the *abdomen*, to the inside of the left flank, 0.12 gramme of *chloride of sodium*, dissolved in 2.4 c.c. of water. The fæces passed previous to the experiment were observed to be firm.

There was a large evacuation during the night, partly firm and partly soft, although not liquid. One day later the fæces returned to their usual consistence.

There was slight swelling at the seat of the injection.

*Experiment XC.*—Same cat. Injected over the outer side of the thigh of the right *fore-leg* 0.125 gramme of *sulphate of magnesia*, dissolved in 2.5 c.c. of water.

There was no evacuation until the second night, when there was passed a small quantity of firm fæces.

Slight swelling at the seat of injection.

*Experiment XCI.*—Same cat; a few days afterwards. At 5 p.m. injected over the *abdomen*, to the inner side of the left flank, 0.12 gramme of *sulphate of magnesia*, dissolved in 2.4 c.c. of water.

During the night there was a large evacuation of fæces, half of which was firm and the other half much softer than usual, although not actually fluid.

There was not much visible inflammation at the seat of injection, although some thickening of the subcutaneous tissue could be felt. The pain on pressure seemed to be inconsiderable.

The chloride of sodium has again relaxed the bowels when injected over the abdomen; and a similar effect has followed the injection of the sulphate of magnesia in the same situation. On the other hand, the latter salt has not affected the stools when injected over a fore-leg. The degree of local irritation caused by the injection was not great, and was much alike in each of the three experiments.

*Experiment XCII.*—Cat, female, weighing 2.29 kilogrammes. Injected over the outer side of the thigh of the left *fore-leg* 0.1 gramme of *sulphate of magnesia*, dissolved in 2 c.c. of water.

No fæces during the next forty-eight hours—hard when they did appear.

Without there being much swelling at the point of injection, the

animal seemed to suffer some pain when the part was pressed or rubbed.

*Experiment XCIII.*—Same cat ; three days later. Injected over the *hypogastrium* 2 c.c. of a solution of *sulphate of magnesia*, containing 0.1 gramme of the salt.

The cat, which, without its being perceived, had been in an early stage of pregnancy, aborted in the course of the night. The consistence of a small quantity of *fæces*, passed towards next evening, was softer than usual, but barely so much so as to be semi-fluid.

The injected part was somewhat swollen and painful.

The experiments on this cat, which in other respects present the same results as those on the previous cat, possess the additional interest of showing that the abdominal subcutaneous injection of a salt may cause the expulsion of the contents of the uterus, as well as those of the intestines, and probably by the local irritation of the injection, acting on the uterus through the agency of a reflex mechanism similar to that which we have supposed to exist between the skin of the abdomen and the intestines.

*Experiment XCIV.*—Cat, female, weighing 2.09 kilogrammes. Injected subcutaneously over the outer side of the upper part of the right *fore-leg* 0.1 gramme of *sulphate of magnesia*, dissolved in 2 c.c. of water.

During the afternoon of the following day there was passed some solid *fæces* of ordinary consistence.

*Experiment XCV.*—Same cat ; two days afterwards. At 7.10 P.M. injected over the *abdomen* 0.1 gramme of *sulphate of magnesia*, dissolved in 2 c.c. of water.

During the night there was an evacuation of a moderate quantity of soft semi-fluid *fæces*. On the following night more soft *fæces* were passed. By the third night the stools had resumed their usual consistence.

There was a little swelling and pain at the seat of the injection.

These two further experiments with sulphate of magnesia are, in their results, in harmony with those that precede, and leave little room for doubt as to the correctness of the theory I have advanced.

I have already shown that even chloride of sodium, a salt which forms the principal part of the ash of the blood, will cause purgation when injected over the abdomen, if its injection is at the same time followed by some degree of local swelling and inflammation. The quantity injected was so small in

comparison with the amount of the salt normally present in the blood, that it is impossible to believe that the absorption of this minute quantity could exercise any effect upon the intestines. Purgation must have been the reflex result of the local subcutaneous irritation.

That purgation, both in the case of the chloride of sodium and of the purgative salts, is produced by such irritation, some experiments with sulphate of zinc very fully support. Sulphate of zinc in small doses is well known to act as a powerful astringent, causing constipation in preference to purgation. Administered by the mouth to cats in doses of from 0.05 to 0.20 gramme, there was either no visible effect upon the stools, or, for a day or two afterwards, the stools were less frequent than usual, and their consistence was somewhat increased. When similar doses were injected subcutaneously over the legs, a like result was obtained. But, when the salt of the injection was the subcutaneous tissue of the abdomen, a laxative condition of the bowels always followed, although, in the two experiments instituted, more tardily than when sulphate of magnesia was employed. The details of these experiments follow,

*Experiment XCVI.*—Cat, female, weighing 2.49 kilogrammes. Injected subcutaneously over the *abdomen* to the inner side of the left flank, 0.5 c.c. of a 10 per cent. solution of *sulphate of zinc*, or 0.05 gramme of the salt.

The injection was made at noon, and by the same hour on the following day no *fæces* had been evacuated, although a slight swelling was visible at the seat of the injection. In the course of the evening of the same day there was a stool, which, although considerably softer than usual, was barely semi-fluid. The local swelling had not increased.

*Experiment XCVII.*—Same cat. Five days after the previous experiment a similar injection was made over the outer side of the left fore-leg, but without any laxative effect upon the bowels.

*Experiment XCVIII.*—Cat, female, weighing 2.09 kilogrammes. Injected over the *abdomen* to the inner side of the left flank, 1 c.c. of a 10 per cent. solution of *sulphate of zinc*, or 0.1 gramme of the salt.

Not until during the second night afterwards were any *fæces* evacuated, when there was a large semi-fluid, dark-coloured stool.

There was a considerable area of soft inflammatory swelling around the point of injection, which continued for a few days afterwards, without that the stools were again semi-liquid.

*Experiment XCIX.*—Same cat. After the swelling over the abdomen.

had completely subsided, a similar injection to that in the foregoing experiment was made over the *back* between the scapulæ.

There were no fæces for two days afterwards, and the first evacuation that occurred consisted of perfectly firm fæces.

These experiments, therefore, bear exactly out what I have already predicated for them. The slowness in both instances with which the laxative effect followed the abdominal subcutaneous injection of the salt may be attributable to the salt by its absorption exercising its ordinary astringent impression upon the intestines, and thus delaying their evacuation until the salt has been eliminated from the circulation.

These now numerous experiments appear so clearly to establish the view I have suggested for the explanation of the action of minute doses of purgative salts subcutaneously injected, that it may be thought unnecessary that I should add to their number by giving a few experiments which were made on man. The only, yet perhaps sufficient, plea I can advance for proposing such an addition is, that it was on man that the first experiments on this subject were made by Luton, and that it was also on man that, later, Gubler performed the very large number of experiments which led to his rejection of Luton and Vulpian's explanation of their results. It became interesting, therefore, to know what effect the salt might possess when attention was paid to the locality into which it was injected. As the operation involved was of the simplest and most harmless nature, some of the experiments were made on convalescent patients within the wards of the Royal Infirmary; and for the performance of these I am indebted to Dr. Hosack Fraser, one of the assistants to the professors of clinical medicine. The remainder of the experiments were made on healthy individuals, and in every instance the person was of the male sex. In every experiment, unless where otherwise mentioned, 1 decigramme of sulphate of magnesia, dissolved in 1 c.c. of water, was injected. In order to test Gubler's theory, some of the patients were told that purgation was expected to follow the injections; the others were absolutely ignorant of the intention of the experiment.

In several cases the salt was injected beneath the skin of the arm; and in no single instance did purgation occur, although in the majority of the cases the individual was led to understand

that such a result was expected. Very rarely was any trace of irritation perceptible at the seat of the injection. It is not necessary that I give further details of these experiments.

As to the results of the experiments when the salt was injected beneath the skin of the abdomen, sometimes purgation followed, and at other times there was no effect. But, as a general rule, it was observed that, when the bowels were affected, the degree of local subcutaneous irritation was greater than when the stools were not softened. Most frequently in the latter case evidence of irritation was entirely absent. The following are the protocols of these experiments:—

*Experiment C.*—Sulphate of magnesia injected half an inch below the umbilicus. Patient not informed of the expected effect.

There was no purgation, and even no apparent acceleration of defæcation.

There was no trace of pain or swelling at the seat of injection.

*Experiment CI.*—Same in all respects as the preceding, and with exactly similar results.

No purgation, and no local pain or swelling.

*Experiment CII.*—Same as the previous two, but individual told that purgation was expected.

Results exactly as before; no laxative effect, and no local pain.

*Experiment CIII.*—Same conditions as in last experiment, and followed by precisely the same results.

*Experiment CIV.*—Same as previously, but individual not informed of the object of the injection.

On this occasion there was some degree of irritation and uneasiness at the seat of injection, and a slight laxative effect on the bowels made itself apparent next morning.

*Experiment CV.*—Same as in preceding experiment, but salt injected into the left iliac region.

A moderate amount of purgation occurred next morning, and there was some irritation at the locality of the injection.

*Experiment CVI.*—Similar in all respects to the last, and followed by much the same results.

*Experiment CVII.*—Injected below the umbilicus 0·05 gramme of sulphate of zinc, dissolved in half a cubic centimetre of water.

In the course of next morning there were two evacuations, both of them soft, but particularly the second, which was fluid.

There was an area of redness, more than an inch in diameter, round the point of injection.



## SUMMARY OF THE EXPERIMENTS OF SERIES F.

No. of Experiment.	Animal.	Salt Injected.	Situation of Injection.	Effect on Part Injected.	Effect on Bowels.
LXXXIX.	Dog.	Sulphate of soda.	Back.	No inflammation.	No purgation.
LXXX.	do.	do.	Abdomen.	do.	do.
LXXXI.	do.	Chloride of sodium.	do.	do.	do.
LXXXII.	do.	Sulphate of magnesia.	Fore-leg.	do.	do.
LXXXIII.	do.	do.	Abdomen.	do.	do.
LXXXIV.	do.	do.	do.	do.	do.
LXXXV.	Cat. A.	Sulphate of soda.	Back.	do.	do.
LXXXVI.	do.	do.	Abdomen.	do.	do.
LXXXVII.	Cat. B.	Chloride of sodium.	do.	Slight inflammation.	Pulpy stool.
LXXXVIII.	do.	Sulphate of soda.	Back.	do.	No purgation.
LXXXIX.	Cat. C.	Chloride of sodium.	Abdomen.	do.	Stool, partly soft.
XC.	do.	Sulphate of magnesia.	Fore-leg.	do.	No purgation.
XCI.	do.	do.	Abdomen.	do.	Stool, partly soft.
XCII.	Cat. D.	do.	Fore-leg.	Slight pain.	No purgation.
XCIII.	do.	do.	Abdomen.	Slight inflammation.	{ Stool, partly soft. (Abortion.)
XCIV.	Cat. E.	do.	Fore-leg.	No inflammation.	No purgation.
XCV.	do.	do.	Abdomen.	Slight inflammation.	Semi-fluid stools.
XCVI.	Cat. F.	Sulphate of zinc.	do.	do.	Soft stool.
XCVII.	do.	do.	Fore-leg.	do.	No purgation.
XCVIII.	Cat. G.	do.	Abdomen.	Inflammation.	Semi-fluid stools.
XCIX.	do.	do.	Back.	Slight inflammation.	No purgation.
C.	Man.	Sulphate of magnesia.	Abdomen.	No inflammation.	do.
CI.	do.	do.	do.	do.	do.
CII. <sup>1</sup>	do.	do.	do.	do.	do.
CIII. <sup>1</sup>	do.	do.	do.	do.	do.
CIV.	do.	do.	do.	Slight uneasiness.	Slight laxative effect.
CV.	do.	do.	do.	Some irritation.	Moderate purgation.
CVI.	do.	do.	do.	do.	do.
CVII.	do.	Sulphate of zinc.	do.	Inflammation.	Purgation.

<sup>1</sup> Individual informed that purgation was expected.

A careful and unbiassed examination of these and the other experiments of this series amply prove, I venture to think, the propositions with which I started,<sup>1</sup> that the salt will not purge

<sup>1</sup> Page 64.

unless when injected over the abdomen; and that, even there, it will fail to act unless its injection is accompanied by a certain degree of irritation; and that its action depends, almost for certain, wholly upon such irritation, independent of the purgative quality of the salt.

In comparing the results of my experiments with those of Luton and Vulpian and Carville, it is somewhat remarkable that, even when I injected the salt over the abdomen, the bowels were by no means always affected, as seems to have happened in the experiments of these other observers; and, when they were affected, the purgation was of a very mild character, the stools being rarely more than partially liquid or semi-liquid. Indeed, had it not been that never once was an injection beneath the skin of the arm followed by the slightest laxative effect, I would have felt inclined to attribute the purgation occasionally observed after the abdominal injection to adventitious causes, and would have maintained that the subcutaneous injection *per se* of saline purges in any part of the body is absolutely without effect on the intestines. The only visible cause of purgation was the irritation which the salt produced at the seat of its injection. When this was entirely absent, no purgation was observed. At the same time, I am not prepared to assert that purgation or a laxative effect will always occur when irritation is visible; probably not. My own experience bears me out in this statement, as do also some experiments by Vulpian. There is, indeed, the possibility that, when the local irritation is intense, an opposite effect is produced upon the intestines, or, at any rate, that catharsis is not induced; for Vulpian on one occasion injected as much as 10 grammes of sulphate of magnesia, dissolved in 20 grammes of water, under the skin of the left flank of a dog; and in another dog, in the same situation, 4 drops of croton oil, dissolved in 10 drops of olive oil. In neither case was there any effect on the intestines, notwithstanding that in both animals there ensued extensive inflammation of the subcutaneous cellular tissue.

It may not be altogether unprofitable to inquire by what means, or through what mechanism, the subcutaneous irritation contrives to cause purgation. The mechanism, as I have already suggested, cannot well be other than nervous and reflex. The

abdominal wall has no direct organic connection with the viscera which it covers. The subcutaneous irritation must produce such an impression on the sensory nerves of the abdominal wall as will be conveyed to some centres in the spinal cord or encephalon in immediate relation with other nerve centres, whose efferent fibres pass to the intestines, and through which the impression will be reflected to these viscera. So much is tolerably certain. But whether the efferent nerves are such as control the secretion of the intestines, or are those which regulate peristalsis, or, perhaps, are merely the vasomotor nerves of the gut, my experiments do not afford me much help in deciding. The stools were not sufficiently fluid as to lead me to believe that secretion was much, if at all, affected. Their character more inclined me to think that it was the motor nerves that were influenced—not only of the intestines, but also of the uterus (Experiment XCIII.). But Carville's observation of a highly congested condition of the intestines in dogs killed shortly after the abdominal subcutaneous injection of sulphate of magnesia, to which I have already referred,<sup>1</sup> renders it probable that it is the vasomotor nerves which are inhibited, and give rise to a temporary catarrhal condition of the intestines. This probability is strengthened by common experience in other departments of medicine, in which we are not unacquainted with an intimate sympathy existing between parts, superficial and deep, totally unconnected by nerves or vessels passing directly between them, and where irritation of the former or superficial or cutaneous part may lead to a vasomotor disturbance of the latter, resulting in congestion and even in inflammation. I need but adduce the instance of the lungs and the skin of the thorax. Hardly any one doubts that congestion and inflammation of the former may sometimes arise from stimulation of the latter by cold or damp; and it has been occasionally observed that a burn or scald of the skin of the thorax has been followed by congestion and inflammation of the underlying portion of the lung.<sup>2</sup>

In citing the instance of the sympathetic relation of the thorax and the lungs, and its affection by cold, as comparable to what occurs when the irritation of an abdominal subcutaneous

<sup>1</sup> Page 63.

<sup>2</sup> Spence, *Lectures on Surgery*, 2nd edition, p. 176, and others.

injection disturbs the vasomotor nerves of the subjacent viscera, I am aware that many physicians would maintain that, in the former case, the application of cold to the skin of the thorax does not produce congestion of the lungs through a reflex nervous mechanism consisting of the cutaneous nerves of the thorax and the vasomotor nerves of the lungs. They believe that the cold causes contraction of the cutaneous arterioles, and, driving more blood into the visceral circulation, produces a purely dynamical and compensatory expansion of the arteries of the subjacent lung, no other mechanism being involved than the blood-vessels and the circulation. This, however, is in the highest degree improbable. For, if the volume of the cutaneous circulation be diminished, and that of the visceral circulation correspondingly augmented, why should the lungs alone be congested, and the remainder of the internal organs remain uncongested? And, indeed, it is very problematical, if congestion of any internal organ, as we ordinarily understand it, can be caused by a rapid, and much less a gradual, increase of the volume of its blood-supply within such limits as are likely or possibly to occur; for physiology teaches us that, owing to the great dilatibility of the veins, both trunks and branches, the capacity of the circulatory system is capable of undergoing sudden and most extensive variations without visible congestion or inflammation of any organ being produced. Were it otherwise, congestion and inflammation of organs would be of frequent occurrence after the application of an Esmarch bandage to the whole length of a lower limb in so-called "bloodless" surgery; and simple transfusion of blood might be attended with the gravest results. Irritation, therefore, of the skin, be it produced by cold, burns, or subcutaneous injections, causes congestion of certain internal, generally subjacent, viscera, not from any disturbance in the equilibrium of their blood-supply acting through the circulation, but from stimulation of a nervous mechanism, which, through the central nervous system, brings the viscera into close union and sympathy with certain cutaneous areas, and through which mechanism vasomotor paresis of the viscera may occur. Thus the abdominal subcutaneous injection of a solution of sulphate of soda, sulphate of magnesia, chloride of sodium, or sulphate of zinc, in proportion to the nature and degree of the local irritation

it produces, can cause congestion of the intestines and consequent diarrhoea; whilst, if it be injected in some region of the body, between which and the abdominal viscera there exists no intimate nervous connection, the injection, however it may affect other organs, will exert no reflex action on the vasomotor supply of the intestines.

Vulpian's experiments render it probable, as I have already remarked,<sup>1</sup> that a very strong irritation of the abdominal cutis does not produce upon the intestines the effect of a weaker irritation. The former may, by exciting a larger area of nervous centres in the brain or spinal cord, bring into play efferent fibres, other than the vasomotor inhibitory, which may nullify the action of these; or, without assuming the involvement of more nerve centres and fibres, it is possible, from the consideration of other physiological and pharmacological phenomena, that an intense stimulation of the same nervous mechanism may have the opposite effect of a weak stimulation.

The explanation which I have offered of the purgative action of a salt when injected subcutaneously is of considerable interest in connection with an observation by Köhler in his elaborate paper on the action of elaterium.<sup>2</sup> Zwicke, one of his pupils, he remarks, had found that injection of an alcoholic solution of convolvulin under the skin of the back, or directly into the vena jugularis, was not capable of purging; injected, however, under the skin of the abdomen, purgation constantly followed. This difference in the behaviour of the cathartic, Köhler attributes to the solution of the convolvulin, in the latter case, penetrating directly through the thin abdominal parietes by a kind of endosmotic action, and thus reaching and entering the intestine without the intervention of the blood-vessels. In the intestines it came in contact with the bile, and was enabled to exert its usual purgative effect, convolvulin being one of those resinous cathartics to which Buchheim and his pupils first directed attention as producing their characteristic action only when mixed with bile. With all respect for the extensive pharmacological experience of

<sup>1</sup> Page 63.

<sup>2</sup> Köhler, *Archiv f. patholog. Anat.*, Bd. i. s. 379; Zwicke, *Wirk. d. Convolvulins u. Jalappins*, Inaug. Dissert., Halle, und *Berl. Klin. Woch.*, 1870, No. 17.

the late and lamented Köhler, the explanation he offers of convolvulin, when thus injected, is to my mind very unsatisfactory and extremely improbable. Can he ask us to believe that the alcoholic solution of the resin will find some path by which it can wander through the muscular wall of the abdomen, through the powerfully absorbent cavity of the peritoneum, and, finally, through the intestinal wall, without in its course being completely absorbed by the numerous blood-vessels and lymphatics it is forced to pass? A much more likely explanation is that which I have suggested for the action of similarly injected salt; the alcohol and the convolvulin locally irritate the cutaneous nerves of the abdomen, and reflexly inhibit the vasomotor nerves of the intestinal canal. Köhler observed that much the same effects followed variously seated injections of an alcoholic solution of elaterin, as when convolvulin was employed.

If I might present yet another instance of purgation following irritation of the abdominal parietes, it is the ancient and long-practised custom of purging children and others by rubbing the abdomen with an irritant cathartic.<sup>1</sup> Since the introduction of croton oil, it has been largely used for this purpose, and notwithstanding the incredulity of Buchheim<sup>2</sup> and some others as to its action being manifested when thus applied, there is no reason to doubt its being able to purge, as otherwise it is difficult to understand how the practice became at one time so prevalent. It was the apparent impossibility of the absorption of the oil by the skin which led such observers as Buchheim to doubt the capability of its acting *by absorption*, and in this he was probably right. For the most recent investigators of the absorptive power of the skin deny its ability to absorb even the most diffusible substances, provided they are non-volatile.<sup>3</sup> But, according to my view, absorption is not requisite, the irritation caused by the application of the oil to the skin being sufficient to account for purgation. And, while its inunction over the

<sup>1</sup> Mérat et Lens, *Dict. univers. de matière médicale*, art. Purgatifs, t. v. p. 544; Madden (*On Cutaneous Absorption*, Grad. Thesis, Edinburgh, 1838, p. 91) cites various authors as stating that a solution of jalap, rhubarb, or scammony, applied to the abdomen, was frequently followed by purgation.

<sup>2</sup> Buchheim, *Arzneimittellehre*, 3 Aufl., 1878, s. 378.

<sup>3</sup> Vile Röhrig, *Physiologie der Haut*, Berlin, 1876.

abdomen is succeeded by catharsis, I am not aware that its application to the chest in thoracic disease has been observed to be followed by a similar effect, although an equal opportunity is presented for its absorption.

Not only do the experiments of this series furnish a reasonable explanation of the results of other observers working with entirely different purgations, but I claim that the latter even support the view I have derived from a consideration of the former.

The most important objection to the saline cathartics acting by absorption when subcutaneously injected lies not in the results of the experiments of this series, but in those of the preceding series, where it was fully demonstrated that a saline cathartic is not capable of purging when injected into the blood.

*(To be continued.)*

A HITHERTO UNDESCRIBED FRACTURE OF THE  
ASTRAGALUS. By FRANCIS J. SHEPHERD, M.D., C.M.,  
M.R.C.S. Eng., *Demonstrator of Anatomy, McGill University,  
Montreal; Surgeon to the Out-Patient Department of the  
Montreal General Hospital, &c.*

LAST year I exhibited to this Society<sup>1</sup> a specimen of fracture of the astragalus found in a subject in the dissecting-room. Since then I have examined the condition of the astragalus in every subject dissected, and have been fortunate enough to obtain two more examples of the same fracture. From this I infer that it is by no means uncommon. The fact that this fracture is not mentioned in any of the text-books on surgery or special treatises on fractures would easily be accounted for by its only being discovered by dissection; it causes no deformity, and the symptoms it would give rise to during life would probably be obscure. In all three examples the fractured portion is the same, viz., the little process of bone external to the groove for the tendon of the flexor longus hallucis muscle. This process is on the posterior border of the astragalus, and overhangs the os calcis. To it is attached the posterior fasciculus of the external lateral ligament of the ankle-joint, called sometimes the posterior peroneo-tarsal ligament. Judging from the appearance of the fracture, it would seem that the process of bone is torn off by the ligament being put on the stretch in some twist of the foot.

In the first case the fractured portion of the astragalus is somewhat larger than in the other two, and is united to the main portion of bone by fibrous tissue and periosteum. It occurred in the right astragalus of a young man, aged about twenty-five years.

The second example of this fracture also occurred in a young man, and in the right astragalus; but in this there is no fibrous or other union, and the broken process is attached to the posterior peroneo-tarsal ligament. It is displaced slightly outwards, and some strands of periosteum pass between its upper surface and the main bone. The broken fragment is quite movable.

<sup>1</sup> A paper read before the Medico-Chirurgical Society, Montreal, April 14, 1882.



In the third case the whole process is not broken off, but only a part of it. As in the first case, the fragment is united with the main bone by fibrous tissue. This specimen was found in the left astragalus of a woman, aged about sixty, whose bones had undergone fatty degeneration.

In all three cases there was no history, and no deformity was made out before dissection. The joints were perfectly healthy looking.

Supposing that this fracture was produced by some twist of the foot, I performed a number of experiments on the cadaver to endeavour to find out the cause. By flexing the foot, the posterior fasciculus of the external lateral ligament is put on the stretch; the tension is still greater when the foot is flexed and twisted out. In every case where this manœuvre was performed I failed, even when the greatest force was used, to break off the little process of bone mentioned above. If the subject was old, and the bones porous, the tip of the internal malleolus was torn off, and if the action was continued, the fibular attachment of the external lateral ligament. If the subject was a young adult, the tip of the internal malleolus was not broken, but the internal lateral ligament was torn away from it; and if the action was continued, the external lateral ligament was torn away from either its fibular or astragaloid attachment. The process of bone external to the groove for the flexor longus hallucis was never broken off, but always remained intact. Thinking that perhaps the fracture might be caused by jumping from a height, and landing on the heels with the foot flexed, and perhaps twisted out, I, in order to simulate this (imperfectly, I must admit), sawed several limbs across below the knee, and, flexing the foot applied force from above by means of a large mallet. Out of five trials I did not succeed once in fracturing the process to which the posterior peroneo-tarsal ligament is attached, but once fractured the sustentaculum tali of the os calcis. That I was unable to produce this fracture does not disprove that it might be caused by a twist of the foot in jumping or otherwise, as I could but very imperfectly imitate the accident as it would occur during life. The sudden twist of the foot, and the force of the weight of the body, with its great leverage, would be difficult to imitate.

Whilst pursuing my investigations in regard to this subject, I

was struck by the fact that the process on the posterior border of the astragalus varies very much in different bones. In some it is almost absent, whilst in others it is very well marked, sometimes overhanging the os calcis considerably, and measuring three-fifths to half an inch in length. The attachment of the posterior fasciculus of the external lateral ligament also varies in extent; in some cases the whole ligament, or one strong strand of it, is attached to this process; in others it is attached to quite a large portion of the posterior and external border as well. This variety in the process and attachment of the ligament would, of course, influence greatly the production of the fracture. I also noticed, that when the groove for the tendon of the flexor longus hallucis muscle was deep the process was prominent, and *vice versa*.

May not this fracture account for some cases of sprained ankles which are so slow to recover, and which occasionally leave permanent lameness, or at any rate weakness? In such cases as I have described, it is probable that any motion of the foot (as flexion and twisting out) which puts the posterior peroneo-tarsal ligament on the stretch would be painful. Some may say that this is not a fracture at all, but merely an example of an ununited epiphysis, as is seen occasionally in the acromion process of the scapula. I have thought of this, and discarded the theory for the simple reason that in the astragalus there is only one ossific centre, viz., in the body of the bone. I have never yet seen, in the numerous examinations I have made of the astragalus in new-born children, a special centre for this process. Again, if it were an epiphysis, there would of necessity be a layer of cartilage between this process and the main bone. Dr. Ino Neill, in the *American Journal of Medical Science* of 1849, describes a fracture of the posterior extremity of the astragalus found in a dissecting-room subject. In this case, however, there was great deformity, with dislocation of the anterior portion forwards; the tibia was forced down between the fragments, and greatly separated them. The fractured portion consisted of the whole posterior border, and included the groove for the flexor longus hallucis.

*Note.*—Since writing the above, an astragalus (right) has come into my possession, in which the process on the posterior border has been broken off, and bony union has taken place.

## A SECONDARY ASTRAGALUS IN THE HUMAN FOOT. By Prof. W. TURNER, M.B., F.R.S.

THE occasional existence of a distinct ossicle, representing the posterior border or surface of the astragalus, which has been described as due to a fracture of that bone by Dr. Shepherd, in the immediately preceding communication, is not unknown to anatomists.

I have now before me the bones of the left ankle, obtained two sessions ago in the dissecting-room of the University of Edinburgh, in which the posterior part of the astragalus, situated behind the articular surface of the tibia, was quite distinct from the rest of the bone. This ossicle was  $\frac{7}{8}$ ths inch in its greatest transverse, and  $\frac{4}{5}$ ths inch in its greatest vertical, diameter. It was marked by a shallow groove posteriorly for the tendon of the flexor longus hallucis. Anteriorly it was smooth and convex, and articulated with the posterior part of the astragalus proper, which possessed a concave surface to which it was adapted. Its inferior surface was smooth, and possessed an articular surface, for the posterior part of the superior articular surface of the os calcis. The posterior division of the triradiate external lateral ligament of the ankle joint was attached to its outer angle. In its anatomical relations this ossicle closely corresponded to the specimens described by Dr. Shepherd.

In the same foot a sesamoid bone was developed in the tendon of the tibialis posticus, close to its insertion into the tubercle of the scaphoid bone.

I first became acquainted with the occasional existence of the posterior part of the astragalus as a separate ossicle by the perusal a number of years ago of an article by Prof. Wenzel Gruber, of St. Petersburg, in the *Archiv für Anatomie und Physiologie*, 1864, p. 286, entitled "Vorläufige Mittheilung über die secundären Fusswurzel-Knochen des Menschen." He describes the ossicle as a "Talus secundarius," and states that, as a rule, it possesses the shape of a quadrant of a sphere. Its size varies, his largest specimen being 10 lines in the transverse, 8 lines in the vertical, and 6 lines in the sagittal diameter. In

the same *Archiv* in 1869, p. 108, Dr. Ludwig Stieda of Dorpat contributes an article on secondary tarsal bones, and describes a similar secondary astragalus in the left foot of a man. Both Gruber and Stieda regard this ossicle as due to a modification in development, owing to the astragalus in these cases having two centres of development. Gruber states that he has seen in the astragalus a special ossific centre for the posterior part of the bone, which may either ossify to, or form an epiphysis distinct from, the rest of the bone. In the latter case, it may either be united to the astragalus proper by synchondrosis, or connected with it by a kind of movable joint, owing to the disappearance of the intermediate cartilage, when it becomes an independent bone.

I entertain a similar view to that held by Professors Gruber and Stieda, and believe that we are to look for the origin of this ossicle, not to a fracture of the astragalus, as described by Dr. Shepherd, but to the presence of a secondary centre of development for the posterior part of the bone, which has not fused with the general body of the astragalus, but has formed an independent ossicle. It is well known that many of both the carpal and tarsal bones, which normally ossify from a single centre, may sometimes possess two centres, each of which forms the nucleus of a separate ossicle, and several instances of these variations in ossification are recorded in the earlier volumes of this *Journal*.

NOTE ON THE *RECTUS ABDOMINIS ET STERNALIS*  
MUSCLE. By G. E. DOBSON, M.A., M.B.

THE pectoral part of the *rectus abdominis et sternalis* extends in most species of Mammalia as far forwards as the first rib, lying between the *pectoralis major* and the ribs. I have lately discovered in the golden moles (*Chrysochloridæ*) a remarkable exception to the usual position of this muscle, for in these animals the *rectus* passes forwards to the first rib superficial to the *pectoralis*, and in close apposition to its fellow of the opposite side, separated only in front by a thin vertical ligamentous partition which connects the longitudinal raphé uniting the cutaneous muscles (*sterno-cuticulares*) with the sternum. This peculiar position of the muscle might appear at first sight to favour the views of some anatomists, that the *musculus sternalis* of man is homologous with the pectoral end of the *rectus* of most mammals.<sup>1</sup>

The position of this muscle in the golden moles is, however, evidently related to the peculiar formation of the thorax in which these animals differ from all other mammals. In them, although they are fitted for fossorial action and underground progression as perfectly probably as the true moles (*Talpidae*), the

<sup>1</sup> These views have been ably disproved by Professor Turner, in his paper on the *Musculus sternalis* in vol. i. of this *Journal*, who remarks that this muscle "approaches so closely in many of its characters to the *panniculus carnosus*, that it may perhaps be regarded as an additional rudiment in man of that very important tegumentary muscle, though it must be admitted that the human platysma lies on a plane superficial to the fibres of the *sternalis* in those individuals in whom they exist." This difficulty may, I think, be got over if we regard the *musculus sternalis* as homologous, not with the *panniculus carnosus* lining the integument, but with the *sterno-cuticularis* (*sterno-facialis* of the hedgehog), which is found in many species of mammals springing from the sternum (extending in some even as far down as the sheath of the *rectus*), and attached to the deep surface of the *panniculus carnosus* either in front of or behind the anterior extremities. The very variable condition of the anterior attachment of the muscle in man also agrees well with this hypothesis, for the rudimentary muscle having ceased to have any function, and having lost its connection with the feebly developed representative of the *panniculus carnosus*, attaches itself irregularly anywhere, though the position of its origin remains to a great extent unchanged.

necessary modifications of the body and limbs have been brought about in a totally different manner. In order to admit of fossorial action and progression in a confined space, the limbs must be so placed that they project as little as possible beyond the sides of the body, while the normal length of the forearms is preserved and the leverage of the muscles remains unaffected. This is effected in the true moles—(1) by change in the position of the limbs caused by anterior elongation of the manubrium sterni carrying with it the clavicles, and (2) by shortening of the clavicles, which is here carried out to the greatest extent known; the limbs being thus brought opposite the narrowest part of the body, and, as it were, articulated with the sternum from which the small quadrate clavicles alone separate them. In the golden moles, however, the manubrium sterni is not anteriorly elongated, neither are the clavicles shortened; but this is made up for by a deep hollowing out on either side of the antero-lateral walls of the thorax, the ribs in these parts and the sternum being convex inwards, the long clavicles have their distal extremities pushed forward, and the concavities on the sides and inferior surface of the thorax lodge the thick muscular arms. The space for origin of the *pectoralis major* is thus much reduced in width, and the muscle itself is closely appressed to the ribs by the superincumbent muscular mass of the arm, leaving no room for the pectoral part of the *rectus*, which, moreover, would have to describe a deep curve if it maintained its usual relations in passing to its insertion. The natural result has followed; this part of the muscle, being put out of action by the altered condition of the thoracic wall, has disappeared along its normal tract; but, evidently in consequence of being essential to the well-being of the animal, has assumed a new course, an occurrence far from uncommon in the muscles of the variable extremities, but much less frequently noticeable in the trunk, and, apparently, hitherto unrecorded in the case of the muscle under consideration.

ECTOPIA VESICÆ AND OTHER IMPERFECTIONS OF  
DEVELOPMENT IN A NEW-BORN INFANT. By  
FRANCIS OGSTON, Jun., M.D., *Assistant to the Professor of  
Medical Jurisprudence in the University of Aberdeen.*  
(PLATES II. and III.)

ABOUT the middle of the winter session of 1880-1 Mr W. S. Lunan, one of our students, brought to the College an infant which had been born in the neighbourhood a few days previously, and which had died soon after its birth.

The child, a male, was about the average length and weight, and, as regards its general appearance, was fat and well formed. On examining it, however, it presented several abnormalities which must be described in detail.

*External Appearances.*—The central part of the anterior wall of the abdomen was occupied by an irregularly triangular smooth patch, of a purplish colour, resembling rather mucous or serous tissue than true skin, exactly, in fact, like the investing membrane of the umbilical cord with which it was continuous, the apex of the triangle being upwards, and a little below the tip of the ensiform cartilage, and its base a little above a line drawn between the two iliac crests. From a point about a third from its apex, sprang an apparently normal umbilical cord (Pl. I. *a*).

Bounding this patch, inferiorly, was a narrow belt of normal skin, varying in breadth from one-twelfth to one-fourth of an inch, and continuous laterally with the skin of the sides of the abdomen.

Underneath the belt of skin a semilunar mass was to be seen, divided into three portions by two vertical depressed lines (Pl. I. *b*), its lateral portions resembling in appearance the patch *a*, and its central portion bright red, velvety, protruding, and plicated. On examining it minutely it was seen to be an everted bladder, with two slit-shaped openings near its upper part (*ureters*), and towards its lower part, on the right side, a wart-like excrescence, about the size of a split pea, apparently the right lobe of the prostate gland; but there was no rudiment of the left lobe.

On exploring the lowest portion of the bladder an opening rather more than a quarter of an inch in diameter was found,

which admitted a probe for some distance; this opening communicated with the rectum.

Below the lateral divisions of the mass *b*, the two halves of a cleft penis appeared, each with a well-formed half scrotum, but of course no trace of urethra (Pl. I. *c*), and underlying them the two divisions of a cleft scrotum (Pl. I. *d*), but no testes could be felt in them.

The raphé of the perinæum showed no rudiment of a rectal opening.

*Internal Appearances.*—In the mouth, throat, and thorax, there was nothing abnormal; but in the abdomen the following abnormalities were found:—

The umbilical cord, on being laid open, was seen to have an umbilical vein, of larger dimensions than usual, and *one* artery which apparently divided at the navel to form the two hypogastric arteries. The umbilical vein penetrated the upper surface of the liver, about a quarter of an inch behind its anterior margin, and then ran its usual course.

On turning up the lower surface of the liver (Pl. II. *g*) it had the usual appearance of division into lobes, but there was *no trace* of the gall bladder or its duct.

The stomach (Pl. II. *a*), was of the normal size, and in the vertical position in which it is found in the foetus; it terminated in the duodenum, which was continued into the jejunum. These portions of the small intestine were about sixteen inches in length, and so far normal that they had a proper mesentery and glands.

The jejunum, however, at its lower end terminated in a somewhat reniform dilated *cul-de-sac* (Pl. II. *c*), about four inches in length, and an inch and a half in breadth at its greatest diameter. This sac was rounded at its upper end, and somewhat pointed at its lower, its upper one-fourth being free, as was its lower half, while between these two parts it was attached by a mesentery about an inch in breadth, with rather large mesenteric glands. At this part the jejunum communicated with it, by gradual enlargement of its canal, and with no semblance of a valve at the point of communication. The sac reminded one of the adult stomach, with the jejunum entering it like the œsophagus, only that there was no pyloric aperture, its lower end being closed. It was half filled with a substance having all the appearance of well-formed meconium.



*The intestine ended here*—no trace of the colon being found.

The rectum, which had been seen to open into the lower part of the bladder, was prolonged upwards for about two inches and a half, terminating in a somewhat constricted blind end. It was lying free in the pelvis, not bound down to the sacrum, and had no connection with the small intestines (Pl. II. *e*).

The spleen (*b*), the kidneys (*d* and *h*), the suprarenal capsules, and the pancreas were normal in size, and in their usual position.

A ureter sprang from each kidney, but instead of terminating in the bladder, ended in a blind end in the subperitoneal tissue at the side of the bladder, at the point where the obturator foramen should have been, having thus no connection with that viscus. (The lines *d*, in Plate I., terminate nearly at the points where they ended.)

The openings in the bladder into what appeared to be the ureters had no connection with the kidneys, but ran up under the peritoneum of the anterior wall of the abdomen, and ended blind—the right one near the umbilicus, and the left one half way to this point.

The right testis had just entered the inguinal ring, the left lay still behind the kidney. The Wolffian bodies were seen behind the kidneys, the left being the larger of the two.

The bony pelvis was deficient anteriorly, the pubic portions of the ossa innominata and the ascending ramus of the ischium being absent.

In the parts *a*, and the lateral portions of *b* (Pl. I.), the abdominal muscles were all but absent, being merely represented by a few shreds of muscle, with hardly any continuous connection, the abdominal integuments there consisting of rudimentary skin and serous membrane (peritoneum).

I neither can nor shall make any attempt to explain this curious case. It would seem as if there had been two if not three interruptions in its development, one affecting the bladder and rectum, with their appendages, giving rise to the defect known as ectopia vesicæ, with epispadic cleft, and cleft penis and scrotum, accompanied by the very usual non-descent of the testes; another affecting the intestinal tract, which appeared to have ended at the point of connection with the yolk sac by the vitello-intestinal duct; and the third, which had given rise to the non-formation of the gall bladder.

NICKEL AND COBALT: THEIR PHYSIOLOGICAL ACTION ON THE ANIMAL ORGANISM. PART I. TOXICOLOGY. By T. P. ANDERSON STUART, M.D., Ch.M., *Assistant to the Professor of the Institutes of Medicine, Edinburgh University.*<sup>1</sup>

THE investigations, of which the present article contains a summary of the results, were carried on during the winter session 1880-81 and the autumn of 1881, in Professor Oswald Schmiedeberg's pharmacological laboratory in the University of Strassburg. I am greatly indebted to him for his kindly encouragement throughout the work. In the same laboratory a series of researches on the physiological action of some other metals on the animal organism had already been carried out by previous investigators. These metals were lead, platinum, mercury, antimony, tin, and iron; and the several accounts of their action will be found in the *Archiv der Experimentellen Pathologie und Pharmakologie*, edited by Professor Schmiedeberg, the Director of the Institute.

Bearing in mind how closely nickel and cobalt are chemically related to each other and to iron, and how constantly it has been tacitly assumed that nickel and cobalt have physiological actions similar to those of iron, it is scientifically a matter of interest and practically of importance to test experimentally the soundness of the assumption.

*Literature.*—The references in medico-scientific literature to the physiological action of the nickel and of the cobalt compounds are at once sparse and meagre, and the details contained therein, as I shall have to show, are not quite trustworthy. They are as follows:—

C. G. Gmelin (*op. cit.*, 1) performed the following experiments referred to by Buchner in 1827. Ten grains crystallised nickel sulphate introduced into a dog's stomach produced merely repeated vomitings, and so did 20 grains given two days later. Ten grains had no perceptible effects on a rabbit, but 20 grains killed a rabbit in a few hours with convulsions, and *p. m.* the gastric mucosa at the cardiac was found to be covered with blackish-red spots. A watery solution of 10 grains injected

<sup>1</sup> This paper is an extract from my Thesis of the above title, presented to the University for the degree of M.D., 1882, and for which a medal was awarded. Part II., which will include the effects of small doses long continued, and the therapeutics of nickel and cobalt, will follow when the investigations are completed.

into the jugular vein of a small dog killed it immediately, and the dissection at once performed showed that the excitability of the cardiac muscle was completely abolished, and that in both sides of the heart there was much fluid blood. Five grains of the same salt injected into the jugular vein of a dog caused in half a minute vomiting, which was repeated from time to time. Diarrhœa set in, and then only a mucous fluid was passed. Soon the dog fell down and lay motionless; in a few hours it began to recover; it remained dull and ill for four days, the apex beat of the heart being weak and slow, and the body greatly emaciated. On the fourth day the eyes were covered with a purulent fluid, but on the sixth day it was well again. 30 and 40 grains of the nickel sulphate laid in the subcutaneous tissue of the neck of dogs were absorbed without vomiting or other remarkable symptom. Thus, says Buchner, we see that the nickel salt only then is particularly dangerous, and by cardiac paralysis rapidly fatal when it is injected into the vascular system; but that also by way of the stomach it may be fatal, death being accompanied by convulsions.

Orfila (*op. cit.*, 1) merely quotes these experiments of Gmelin.

Huseman (*op. cit.*, 3) says that the nickel and cobalt salts agree in their action with the chloride of manganese, and with potassium manganate and permanganate. In acute poisonings with 1 to 3 drachms of the sulphate, he says, there is said to be irritation and corrosion of the intestine, and death with vomiting and convulsion; also great secretion of bile.

Broadbent (*op. cit.*, 4 and 5), assuming that the members of the iron group of metals will have similar physiological actions, proceeds to prescribe manganese, nickel, and even chromium salts in cases where iron is generally employed, *e.g.*, anæmia, menorrhagia, amenorrhœa, and leucorrhœa, and he states that he has seen beneficial results follow.

Buchheim (*op. cit.*, 6) says that small doses of nickel and cobalt salts introduced into the alimentary canal do not induce symptoms of poisoning; and that with regard to the effects of their intra-venous injection we do not yet possess sufficiently exact knowledge.

Azary (*op. cit.*, 7) does not separate the action of the nitrates of nickel and cobalt. In acute cases he found stupefaction and lowering of the temperature; then the heart's action and the respiratory movements slowed and weakened, the pupils dilated. In chronic cases there were emaciation. The urine increased three or four times, of a dark brown colour; briny when cool; sp. gr. may exceed 1049; there may be albumen and hyaline casts. The frog's heart first is slowed, then ceases to beat. In the dog the pulse is first accelerated and the blood pressure rises; then the pressure falls, the pulse slows, and the heart ceases to beat. This is a purely local action affecting its muscle and its automatic ganglia. An affection of the vagi or of the accelerating fibres was not observed. The hæmocytes swell, lose their pigment, and become transparent sacs; in frogs they may fall to pieces.

Sir J. Y. Simpson (*op. cit.*, 8) prescribed nickel sulphate  $\frac{1}{2}$  to 2 grains as a gentle metallic tonic; found that large doses produced sickness and nausea, and believed that nickel and manganese in their action so corresponded with iron, that these three metals might be substi-

tuted the one for the other. Nickel he found completely successful in a case of severe periodic headache where quinine and other medicines had failed, and he thought that he had seen it beneficial in chlorosis.

Palmer (*op. cit.*, 9) prescribed sulphate of nickel as a sedative in painful affections, *e.g.*, neuralgia, or as a soporific where opium was not tolerated, and always successfully. He states that its use is not followed by disagreeable sensations, nor by alimentary derangement.

Buchner (*op. cit.*, 1) states that, according to C. G. Gmelin's experiments, the cobalt salts act in general like the nickel salts. Ten grains of cobalt chloride injected into the stomach of a dog induced only repeated vomitings. Six grains of cobalt sulphate injected into the stomach of a rabbit killed it in a few hours; *p. m.* the gastric mucosa at the cardiac end was studded with dark red spots, and at the greater curvature were broad brownish-red spots; in the lungs were two or three dark red parts of the size of a lentil, and which could not be displaced. Three grains of the sulphate dissolved in 2 drachms of water, and injected into the jugular vein of a small dog, caused in one minute vomiting, several times repeated, and tenesmus also appeared; next day the vomiting recurred, the animal was dull-looking, and from time to time gave signs of pain; the pulse was accelerated; on the fourth day death supervened. On dissection reddened spots were seen in the stomach and duodenum, and the ileum projecting into the colon formed a volvulus, caused perhaps by the continual retching and vomiting during three days. When 6 grains of cobalt chloride were injected into the external jugular vein of a small dog, there followed some deep respirations, the heart's beat became inappreciable, and death supervened within half a minute. This result appeared to depend on a paralysis of the heart. Twenty-four grains of dry chloride of cobalt laid in a wound in a dog's neck appeared to cause much pain, in five minutes some vomiting followed, but the dog gradually got better, and next day was well.

Hasselt (*op. cit.*, 10) states merely that the sulphate and chloride of cobalt agree in their physiological action on dogs and rabbits, and that they have emetic properties.

Huseman (*op. cit.*, 3) says that the salts of nickel and cobalt agree with the chloride of manganese and the permanganate of potash, that  $\frac{1}{2}$  drachm doses of the oxide of cobalt cause the death of dogs in a few hours, and 24 grains of the chloride given endermically cause vomiting. He says that the cobalt compounds are always met with rendered impure by the presence of arsenic, and he ascribes these phenomena, as well as poisonings by Smalt, to the presence of that arsenic.

Siegen (*op. cit.*, 11) employed nitrate and chloride of cobalt absolutely free from arsenic, and found cobalt a poison *sui generis*. 0.01 gramme of the substance killed a frog in half an hour, and 0.3 gramme killed a strong rabbit weighing 1.5 kilogrammes in three hours. The heart's action in the frog falls to half or quarter the normal, and in five minutes it stands in diastole, neither mechanical stimulation nor vagal section now sufficing to restore its action. Thus he assumes paralysis of the cardiac muscle. In the case of rabbits, 0.1 gramme (mode of administration not stated) caused great dyspnoea, and the pulse fell from 178

to 128; after lethal doses death supervened with increasing dyspnoea, the reflex excitability remaining. A rabbit received 0·1 gramme of cobalt (metallic) as chloride, and in the beginning of the poisoning showed great myosis, great dyspnoea, and diminution of the pulse frequency, which symptoms lasted two and a half hours and then completely disappeared.

Buchheim (*op. cit.*, 6) applies the same remarks to cobalt as to nickel. Azary's work before quoted applies evidently equally to cobalt and to nickel.

Huppert (*op. cit.*, 12) almost leads one to infer that the cobaltous salts are not poisonous, for he writes, "nickel and cobalt salts may be absorbed, and the former are poisonous."

In the *Dispensatory* of the United States of America (Philadelphia, 1879), it is stated that an oxide of cobalt prepared by precipitating the chloride with potassa fusa has been employed in rheumatism, that it is emetic in doses of 10 to 20 grains, and that the salts of the metal are irritant poisons.

*Summary of Previous Investigations.*—To sum up all these experiments, those of Gmelin are the most trustworthy and instructive: the sulphate of nickel introduced into the stomach acted as an irritant poison, while introduced into the blood the larger doses caused death by cardiac paralysis, and the smaller doses a train of symptoms which, as I shall show, are in the main correctly given, but which are not followed up sufficiently closely. Most curious is the inactivity of the salt laid in the subcutaneous tissue, for in my experiments subcutaneous injection of the solution was a most excellent mode of administration. Azary's experiments come next. I too have noted the increased quantity of urine, but that is due, I think, merely to the great thirst, and consequently the large amount of water ingested. He mentions the brown colour of the urine and the loss of pigments sustained by the hæmocytes. I have never seen this in my acute cases, even where the urine (in cobalt, not in nickel cases) was almost black; if he has regarded this colour as due to decomposed blood pigment, which it is not, then that may have led to a mistake about the change of the blood-corpuscles. I have never seen the rise of blood pressure, and, as I shall amply prove, the fall of the blood pressure does not depend on the heart. Gmelin ascribes to cobalt chloride pretty much the same action as to nickel sulphate, but the cobalt salt did act from the subcutaneous tissue. Siegen's experiments with cobalt, nitrate, and chloride seem to show that it paralyses the heart. The other authors notice the enteritic effects mostly.

All these experiments have been performed with salts of the metal and a strong mineral acid—sulphate, chloride, nitrate. Such salts in watery solution have a strongly acid reaction, and have a strong affinity for the albumins of the tissues, which they may coagulate, although coagulation is not necessary for the destruction of the constitution of the tissues (*op. cit.*, 13). Thus they may locally destroy tissue, while but little of the metallic compound is absorbed into the system generally. Accordingly many so-called “acute metallic poisonings” are really not specific, but simply the results of the local action of caustic substances.

*Conditions of Experiment.*—In order to ascertain what is the general action of a metallic oxide, the combination in which it is administered

1. Should be readily soluble in watery media at the temperature of the blood.
2. The solution should be neutral or just slightly alkaline, for otherwise the reaction of the blood may be altered and the whole metabolism of the body affected.
3. It should not coagulate proteid substances in neutral or in alkaline solution, nor
4. Should it be precipitated by alkalis nor by the alkaline carbonates, for in these cases it would not only be rendered inert, but might lead to embolism and other conditions, and thus give rise to symptoms altogether apart from the action of the metal.
5. It must not contain any active constituent other than the metallic oxide in question, and thus the strong mineral acids are not admissible, for they have an action of their own.
6. It must not locally excite more than any other soluble and pharmacologically indifferent salt, so that substances with a “caustic” action are clearly unsuited.

Many efforts have lately been made to produce preparations which would satisfy these conditions, *e.g.*, double salts of mineral acids, and sodium with zinc, copper, platinum, and lead respectively. Then also compounds of the metallic oxide with organicbodies, such as mercuric oxide with glycoll, asparagin, and alanin, first prepared by Dessaignes, and employed by

v. Mehring; plumbic oxide with tri-ethyl acetate employed by Harnack, a compound which was not very suitable, for it had an action peculiar to itself as a compound, the lead action supervening later on in the case; ferric oxide as a double salt with sodium and tartaric acid; and various other oxides combined with peptones, and so on. Some of these are suitable, some are not, *e.g.*, the tri-ethyl lead acetate.

In my first experiments I used the double tartarate of sodium and nickelous oxide, but found this unsuitable, since it can be preserved only in acid or strongly alkaline solution. Then I tried a double salt made by dissolving nickelous carbonate in a solution of pyrophosphate of soda, but this proved too weak as a solution of nickel, and I afterwards found that it was strongly active from its pyrophosphatic side. Next a solution of simple nickelous citrate rendered alkaline with caustic soda also soon gave a rich precipitate. Finally, I found the compound which I have used in my experiments. It was first prepared by Heldt. I prepare it thus:—Dissolve two parts of crystallised citric acid in a small quantity of distilled water, and with the aid of heat so as to make a syrupy solution, neutralise this solution with strong caustic soda solution, and to the neutral solution add one part more of the acid and let it dissolve. Dissolve sulphate of nickel in hot water, and precipitate with sodic carbonate. Dissolve the freshly precipitated and thoroughly washed nickelous carbonate in the solution of acid citrate of soda with the aid of heat. The mixture may require to be raised to the boiling point; allow it to cool, and then filter. The filtrate is of a beautiful apple-green colour, and forms a glass when it is evaporated. It may be too alkaline, in which case some citric acid must be added to approximate the alkalinity to that of the blood. It is perfectly stable, and may be administered by all the modes. The cobalt salt is produced in exactly the same manner and has the characteristic purple colour. I have found the same combination suitable for iron and for manganese.

These solutions satisfy my conditions, for they may contain 10 per cent. of the metallic oxide, may be rendered just alkaline, do not precipitate blood serum, nor are they precipitated by alkalis or alkaline carbonates when these are added to them in any proportion. Their sodium and citric acid in a lethal dose of

the compound are in so small amount, and their intrinsic action is so unimportant, that the only active constituent is the metallic oxide. Locally, they may be applied to tongue, muscle, and nerve without causing the slightest irritation.

The chemicals which I used were obtained from Merck of Darmstadt. They were always tested by the Marsh method for arsenic, and were always found to be arsenic free.

The dose is always reckoned as "oxide per kilogramme" animal.

Post-mortem examinations were made in all cases.

The animals experimented on were frogs, fish, pigeons, rats, guinea pigs, rabbits, cats, and dogs, and the experiments numbered about two hundred.

#### *A. General Action of the Salts of Nickel and Cobalt on Frogs.*

The action is best seen when a large dose is injected into the dorsal lymph sac; the phenomena are seen long before all the dose is absorbed, but by the large dose the needful tension of the poison is attained. First the colour of the skin all over the body becomes darker and more uniform, and not unfrequently a white froth like a soap lather is abundantly produced all over the integument. Now follows an interval, often of about twenty minutes, during which the frog sits quite quietly with the eyes retracted and shut. If it be molested now it moves, but in a clumsy manner, and soon comes to rest. In this resting state the fore limbs seem weak, and the hind limbs are drawn up in a most peculiar fashion—the limbs are altogether too much drawn up, the thighs are so jammed up against the body that they come to lie on the dorsal aspect of the sides of the animal, and the legs are so much flexed that the feet lie on the animal's back, quite internal to the planes of the thighs. At this stage the dorsal decubitus is tolerated. Soon some fibrillary twitchings are observed in the muscles of the abdominal wall, then feeble twitchings of the fingers and muscles of the fore limbs generally, lastly the toes are seen to twitch, and then the muscles of the hind limbs generally. This order is always observed. These twitchings become more and more pronounced, and are followed by distinct cramps. Now spasmodic gaping may be observed,



and if the animal is made to move it moves incoordinately; between the cramps the twitchings persist. The symptoms at this stage are extremely like those produced by picrotoxin. In a short time, and suddenly, true and absolute tetanic attacks supervene, in which sometimes emprostotonos, sometimes opisthotonos, is the more marked; now the outward condition is that of strychnine poisoning, and good pharmacologists have not been able to distinguish the two conditions.

The clonic and tonic attacks now cease, and a stupefaction of the animal sets in. In this condition either it will retain the dorsal position indefinitely long, or if it tries to turn round it cannot do so. Seemingly a voluntary motor paresis has set in, while the reflex motion called forth by percussing the toes is decidedly increased. The respiratory movements of the pharyngeal floor are extremely irregular, and the paresis becomes complete paralysis. The heart—not laid bare—beats more and more slowly and feebly, the respiratory movements cease altogether, and death gradually and imperceptibly supervenes. Post-mortem—rigor mortis; at most slight congestion of alimentary tract; heart with the auricles dilated, and filled with dark blood; ventricle mostly small, pale, and semi-contracted. For some time after death the nerve trunks and muscles react to the induction stream.

B. *Experiments to ascertain the influence of Nickel and Cobalt Salts on the function of Striped Muscle.*

Considering the interesting results obtained by Harnack (*op. cit.*, 14) as to the action of lead of copper and of zinc on striped muscle, and considering the statements made with regard to cardiac paralysis as one of the outstanding features of nickel and cobalt poisonings, it became a matter of importance to ascertain the effect of these metals on striped muscle work.

These experiments were carried out with the Kronecker-Tiegel apparatus (*op. cit.*, 15) and with Rosenthal's "Frosch-Caroussel" (*op. cit.*, 16), which is a modified form of Fick's "Arbeit-Sammeler." The method pursued is set forth in minute detail by Kobert (*op. cit.*, 17), but shortly it is as follows: one gastrocnemius of the normal frog is excised, and hanging vertically is

attached by its upper end to a pair of muscle forceps, and by the tendo-achilles to a lever, and this lever is provided with a style at its end, and the style impinges on the smoked enamelled paper applied to the revolving cylinder. Platinum wires, coming from the secondary coil of a Du Bois-Reymond induction machine, are thrust through each end of the muscle. Two Grove's cells are connected with a Bowditch clock, and the clock is usually set so that the current is closed every four seconds. By this closure a bar of soft iron is converted into an electromagnet, and a lever is pulled down, the Kymograph cylinder is permitted to revolve through a small distance, and an induced shock is communicated directly to the muscle, which consequently contracts, and raising the lever makes an ordinate on the smoked paper. A weight is attached to the lever, so that each ordinate means that the muscle has raised that weight through that distance, and the sum of the ordinates expresses the whole work done by the muscle. Ordinates continue to be taken until the muscle is exhausted. By ligaturing the leg before the operation no blood is lost to the frog. The temperature of the room is kept as nearly as possible constant. The frog is poisoned, and when the desired stage of the metal's action is reached, the other gastrocnemius is prepared in the same manner, stimulated by the same strength of shock, at the same intervals of time as in the case of the normal muscle, and thus the work of the poisoned muscle is ascertained. A long series of control experiments show that if both muscles were normal they would yield the same result, although an interval of several hours may elapse before the second gastrocnemius is operated on. Thus it was experimentally proved that to operate on the one gastrocnemius, to poison the frog, and in due time operate on the second muscle, does not impair the results for comparison.

Kebler (*op. cit.*, 18) and von Mehring (*op. cit.*, 19) state that in animals about to die of platinum and mercury poisoning respectively, the muscular excitability is diminished, but by the Kronecker-Tiegel method a diminution may be detected much earlier than they found by the Du Bois-Reymond's coil and simple inspection; it may thus be found in animals that eventually do not die of the poisons. Similar results are given for arsenic by Lesser (*op. cit.*, 20) and for antimony by Soloweitschky

(*op. cit.*, 21). It is the same in the case of copper and zinc and in all these cases as in the normal and in nickel and cobalt, the line joining the summits of the ordinates is straight. The ordinates drawn by a muscle poisoned with lead are of irregular lengths, longer and shorter ones succeeding each other without definite order, and thus the line joining their summits has an undulating course as it falls from the summit of the highest ordinate to the abscissa.

The following table gives the results of some of these experiments.

In 1 and in 2 the poisoned muscle actually yields more than the normal one, due to the higher temperature; and the male frog yields more than the female because of the large size of the ovaries at the time. When we consider the numerous factors influencing muscle work (Kobert, *loc. cit.*), and compare the work done by the poisoned muscles and by the normal muscle of the same frog, or of frogs of the same size and sex, we see that neither nickel nor cobalt have any appreciable effect on striped muscle. Thus they differ from platinum, arsenic, antimony, mercury, lead, and from iron, in large doses—all of these diminishing the work yielded. Small doses of iron increase the muscle work (Kobert).

### C. *Action on the Nervous System of the Frog.*

If the sciatic nerve of one side be severed, and the frog now poisoned, the twitchings and other motor signs appear only on the intact side, and if while the cramps are present the spinal cord be destroyed or the sciatic nerves divided, then they cease. Clearly then they are not due to any excitement of muscle and peripheral motor nerve ending, or motor nerve fibre—they are due to an excitement of the central nervous system. This might be supported, if need be, by these facts, viz., that they always appear in the order given, and that when the artery of the limb is tied previous to poisoning, they appear still. If the encephalon be laid bare, and the frog now poisoned, then when the twitchings and cramps are present, the fore-brain severed from the mid-brain, then the mid-brain from the hind-brain, then the myelon from the encephalon, the motor

TABULATED RESULTS OF SOME EXPERIMENTS TO ASCERTAIN THE EFFECT OF NICKEL POISONING ON MUSCLE WORK.

*Rana Temporaria.*

No.	Condition of the Muscle.	Distance apart of Colla.	Weight raised in Grammes.	Times the load is raised per min.	Leverage.	Temperature. Centigrade.	Duration of Experiment in minutes.	Greatest height to which raised.	Weight of Frog.	Work done in Grm. mm.
1. ♀	Normal,	80 mm.	30	10	2	20°·5	49	6·6	49	25,440
	Within 40 min. ·612 NiO per kilo. frog in 1 % sol. injected into the aorta, . .	"	"	"	"	20°·5 +	45	7·8		27,675
2. ♂	72 hours after ·139 NiO per kilo. frog subcutaneously,	80 mm.	20	15	2	12°·5	45	6·4	38	27,225
	194 hours after that injection, . . .	"	"	"	"	20°·0	48	6·1		28,050
3. ♂	18 hours after ·161 NiO per kilo. frog subcutaneously, .	85	20	15	2	21°·0	55	5·6	31	31,275
	42 hours after ·150 NiO per kilo. frog subcutaneously, .	80	20	15	2	20°·0	40	7·2		26,850
5. ♂	96 hours after ·128 NiO per kilo. frog subcutaneously, .	85	20	15	2	10°·0	43	5·6	39	23,850
	120 hours after ·156 NiO per kilo. frog subcutaneously, .	85	20	15	2	17°·0	40	5·3		25,350

symptoms still persist; thus they are due to an excitement of the myelon. This excitement of the spinal centres is probably succeeded by their paralysis—certainly the muscle remains excitable through the nerve for long after death, and the muscle work experiments show that that is not diminished, and the reflex excitability at first exalted is then gradually abolished.

Whether or not there is any affection of cerebral centres is doubtful. The peculiar attitude and the cramps in the early stage give quite the impression of poisoning by picrotoxin, cicutoxin, toxiresin, and other members of this group (*op. cit.*, 22). Böhm and Mickwitz (*op. cit.*, 23) refer barium (chloride and acetate) to this group. Barium causes a peculiar attitude, but different from the nickel and cobalt attitude—the thigh is extended, the knee is flexed, and thus the soles are applied to each other. Fibrillary twitchings, clonic and tonic spasms, and paralysis of voluntary motion are also here noted. They record also a symptom which I noted in my experiments with nickel and cobalt, namely, a shrill piercing cry, utterly unlike the sounds usually emitted by frogs, and they I think rightly attribute it to simultaneous spasm of the muscles of the abdominal wall of the glottis. Huseman (*op. cit.*, 22) thinks that this cry is due to pain from the enteritis, which he thinks also accounts for the other symptoms in frogs, and thus he does not admit barium as a member of the picrotoxin group. This assumption certainly will not do for nickel and cobalt, for I have never seen good evidence of enteritis in frogs, and in mammals signs of pain are neither frequent nor unequivocal. Huseman regards the convulsions of mammals in barium poisoning as due to cardiac paralysis and asphyxia—that does not apply for nickel and cobalt, and probably not for barium either.

Kebler (*op. cit.*, 18) found for platinum a similar train of symptoms, but describes no peculiar attitude. The muscle substance is affected, although voluntary motion is suspended long before the affection of the muscle substance is very marked. He says that the jerkings and cramps are like those caused by the picrotoxin group. Iron (Meyer and Williams, *op. cit.*, 24) causes in acute poisonings slight motor excitement, followed by paralysis, and tin (White, *op. cit.*, 25) does likewise.

These then are the—in this connection—most recent accounts of the actions of the metallic oxides, and we see that nickel and cobalt have a somewhat peculiar action on the nervous system of the frog, agreeing most closely with platinum and barium, but differing from iron. It is probable that nickel and cobalt do paralyse to some extent the cerebrum, as shown partly by the retention of the dorsal decubitus and by the succeeding stupefaction.

*D. Action on the Blood and on the Vascular System of the Frog.*

I have never noticed any change in the appearance of the red blood-corpuscles of the frog in acute cases of poisoning with nickel and cobalt. Azary states that he has seen the blood-corpuscles of the frog fall to pieces, but then he used a 2 per cent. solution of cobalt nitrate, and a 2 to 5 per cent. solution of nickel nitrate salts, which by no means satisfy the strict pharmacological conditions set forth in the beginning of this paper. When I mix my strongest solutions with blood, no other change is seen in the corpuscles than would be caused by any other indifferent salt.

When the frog's heart is exposed, the pericardium being left unopened, it is seen to beat more and more slowly, to become smaller and paler, and not to contract evenly throughout the extent of the ventricle, but the rhythm of the auricular and ventricular contractions is never lost. When the abdomen is now compressed, or when the frog is simply inverted, the heart swells up, fills with blood, and beats well enough again for a time at least.

Vagal section and atropia prove that there is no excitement of the cardio-inhibitory mechanism. The result of the experiments on the muscle work of the frog, and the powerful cardiac contractions following the filling of the heart with blood, render it extremely improbable that there is any paralysis of the cardiac muscle. Probably there is first a vasomotor paralysis of the abdominal blood-vessels, the blood pressure falls, the heart is not nourished as it should be, nor is the endocardium stimulated by the blood within the ventricle, therefore the heart is small, and its contractions are

fewer and feebler, all the more since the pharyngeal respiratory movements are by this time suspended. It is not likely that the motor ganglia of the heart are seriously affected, because of the heart beating well when it is filled with blood, and because the rhythm is never disturbed.

Of the other metals, the above-mentioned authors state that iron has not any effect on the frog's heart, tin weakens and renders it anæmic from a nervous paresis, mercury slows and makes its action irregular, and may so disturb its rhythm that two auricular contractions precede one ventricular, barium has quite a digitalis-like action, platinum affects the cardiac muscle less than voluntary striped muscle.

#### *E. Experiments on Pigeons and Guinea-Pigs.*

The experiments on pigeons are but four in number, and were performed only with nickel, administered subcutaneously. There is first a dulness and stupor, then feebleness of voluntary movement, jerkings of different sets of muscles, unsteadiness of motion, and death ensues quietly; as far as was noticed, a paralysis of the voluntary motor apparatus preceded it. Post-mortem—rigor mortis well marked, blood normal, cardiac ventricles semi-contracted and pale, some congestion of the intestinal mucosa.

Harnack experimented with lead on pigeons, and found the effects quite comparable to the symptoms in the cases of rabbits and dogs.

On guinea-pigs two experiments were performed with nickel and two with cobalt, the metals being administered subcutaneously. In all cases only a dulness or stupefaction, followed by weakness of the extremities, especially the hind limbs, was noticed. In the nickel cases nothing pathological was found post-mortem. In the cobalt cases some gastric congestion and small hæmorrhages in the mucosa.

#### *F. Experiments on Rats.*

The symptoms manifested after subcutaneous injection of the nickel salt are almost entirely nervous. They become less noisy,

less easily excited, drowsy and apathetic, though there is a certain restlessness withal. After the larger doses a paralysis of the hind legs now appears, and gradually extends over the body, and the respiratory movements become slower and more laboured. The breathing becomes shallow and irregular, the general paralysis completes itself, and death ensues quietly. With the smaller doses this drowsiness and apathy may not be developed, or may pass off again; so complete is the apathy sometimes that fierce male rats may be handled with perfect impunity. During this stage the ears and feet appear decidedly redder. Eventually with smaller doses a stage of motor excitement supervenes, and this may be expressed by irregular movements of the fore legs, by unsteadiness while walking, or by a constant chorea-like tremor of the whole body. In one case, while the animal lay on its side affected with voluntary paralysis, the fore legs moved as if in swimming or in running. In another case there were peculiar spasmodic swayings from side to side; following this motor excitement comes motor paralysis, the congested parts become pale, the temperature of the body falls, and death supervenes as above indicated. Post-mortem examination discloses nothing pathological; the auricles are always distended with dark fluid blood, the ventricles pale and semi-contracted, blood-corpuscles normal.

The experiments on rats with cobalt give a similar train of symptoms to those given by nickel, in one case only diarrhoea was noticed. The dissections showed either nothing pathological, or some vascular injection of the alimentary mucous membrane.

Rats, on the whole, are somewhat unsatisfactory animals to experiment on; and since the results of these experiments are in complete accordance with the results in the case of the larger animals, I shall defer any comment until I speak of rabbits and dogs.

#### *G. Experiments on Rabbits.*

Acute poisoning may be induced through the stomach, or by injection subcutaneously or intravenously. First there is an acceleration but no irregularity of the pulse, the respiratory movements are quicker, often irregular, and not unfrequently



they are extensive, and as if voluntarily deep and laboured. When the injection is directly into the blood, except it be very slowly performed, spasms of the whole body appear, and urine and fæces are discharged; sometimes there is myosis. If the animal be now freed from the holder it appears stupefied, and is paralysed. The paralysis may affect only the hind legs or only the fore legs, or all four—the hind legs lying together, the fore legs thrown outwards each to its own side, so that the animal's breast rests on the ground. The cervical muscles may be so weak that the head is not held up. Diarrhœa now appears, and persists more or less till death. If the dose be very large, the animals may not recover, but simply die without other noteworthy symptom. Short of that, however, recovery from this paralytic state ensues, and now the reflexes are decidedly increased, so that the slightest excitation may cause the animal to cower together and tremble all over. Now appear twitchings and contractions of individual groups of muscles, and this excitement becomes general. Respiration becomes slower and more difficult, and sometimes there is now a well-marked dilatation of the vessels of the ears and fundi oculi. One or more violent convulsions come on, the breathing is the longer, the slower, and the more difficult, and, after a convulsion, death follows. If the thorax be now opened, the heart is found beating well and regularly, and it continues to do so for some minutes.

When the course of the case is subacute or chronic, the paralytic symptoms are less marked, while the excitement symptoms are more marked, more varied in their character, and more persistent, and loss of appetite, emaciation, and anæmia appear.

*Post-mortem.*—Rigor mortis well marked, blood dark cherry red, fluid or coagulated. Almost constantly numerous little extravasations of blood in the gastric mucosa, and also, though less constantly, in the intestinal mucosa. In the stomach they may be confluent, and thus form a great, nearly black area that may be covered by a slimy, dark coloured, blood-stained layer of mucus and debris of tissue, or the spots may have become ulcers covered by a distinct scab. In the intestine the destruction of tissue never went so far, and the continuity of the

mucosal surface was never broken; nothing pathological elsewhere. In certain cobalt cases small extravasations in the pleuræ pulmonales, endo- and epicardium.

The diarrhœa is first the passage of the solid balled fæces already present in the intestine, but soon the fæces become thin and fluid; I have never seen them bloody. This diarrhœa is certainly quite independent of the food, for other animals similarly fed showed none of it. The appetite may remain unimpaired. In one case, after a single dose of nickel administered subcutaneously, it recurred at intervals during a period of thirty days, and it might have persisted longer had not the animal been killed. When Boehm and Mickwitz opened the abdomen, after the injection of barium salt into the blood, they saw the intestine so firmly contracted that it looked like a white cord; to this increased peristalsus therefore they ascribe the diarrhœa. They seem to have seen also the bladder contract. They found also an enormous rise of the blood pressure which they say is due to contraction of the lamina of the smaller vessels; thus barium seems to cause a widespread contraction of non-striated muscle—whether due to intrinsic changes of the muscle substance or to excitation through the nervous system they leave undecided. The same causes probably operate in the case of nickel and cobalt, but only for the first passage of intestinal contents. The subsequent diarrhœa is probably due mainly to increased secretion of fluid by the intestinal mucosa. This is certainly not due to local irritation of the mucosa, because large quantities of the salt introduced *per os* may have little effect, and the quantity of nickel or of cobalt secreted by the mucosa though appreciable is yet very small, and the bile is not remarkably increased in quantity, nor is its quality altered, except that it contains a small quantity of the metal. Undoubtedly a gastro-enteritis develops itself probably through some unexplained action of the nervous system. Also in acute tin, lead, platinum, arsenic (Unterberger, *op. cit.*, 26), and iron poisonings, and after mercury given subcutaneously diarrhœa is noticed, and in platinum cases it may be bloody. Kebler, speaking of platinum, thinks that the enteritis is sufficiently accounted for by the extreme dilatation of the abdominal blood-vessels. This explanation does not suffice

for nickel and cobalt, for I have frequently seen marked diarrhoea where there was no such vascular dilatation, and but trivial local changes in the mucosa even.

The nervous symptoms are altogether very complex. The tonic and clonic cramps that are constantly observed on somewhat rapidly injecting the poison into the blood directly are noted in barium poisoning too, and are certainly of purely nervous origin. They are not due to an asphyxic state, as the blood pressure experiments show. The experiments on frogs, the decidedly nervous symptoms in subacute and chronic cases, the non-occurrence of the asphyxic state, and the after-coming paralysis, all point to the spinal cord as the seat of origin of these initial cramps.

The affection of the spinal cord is the outstanding feature in the cases. The reflex excitability is enormously increased, so that pinching the foot of the animal, or even stamping one's foot heavily on the floor, may call forth a general tremor or a violent convulsion. Spontaneously there may be merely a tremor or shiver all over, or the movements may be such as to resemble paralysis agitans of man. In one case (a dog) the picture was entirely that of chorea in man. Particularly towards the end of the case the contractions become more violent and extensive, and now perfect tetanic attacks may supervene. In the intervals the animal is the subject of complete voluntary paralysis. The cramps are not due to asphyxia, since the respiration and circulation may be quite unimpaired when the cramp suddenly comes on. Sometimes cries are uttered, may be due to pain, but usually to simultaneous spasm of the muscles of the glottis and of the trunk.

Consciousness seems to be retained during the paralysis, as Harnack found also in his lead experiments. Sometimes there seems to be a psychical exaltation as noticed by v. Mehring in mercury poisoning, and by White in tin poisoning (*loc. cit.*).

A gravid animal aborted after six daily doses of  $1\frac{1}{2}$  mgr. nickel oxide subcutaneously, all the young being dead-born. Two other cases showed the peculiar so-called manège movements.

### H. *Experiments on Cats and Dogs.*

Since I have not noticed any notable difference in the symptoms of nickel and of cobalt poisonings of cats and dogs, it is convenient to group these experiments under one heading.

When the solutions are injected directly into the blood, and particularly when the injection is somewhat rapidly carried out, there is here, as in rabbits, a general motor excitement. With this, or following closely upon it, the urine and fæces are discharged, and violent retching and vomiting appear. When the injection is slowly performed, or when the dose is administered subcutaneously, this motor excitement is not seen. When the animal is freed from the operation table, either it lies down exhausted, or stands still a while as if it did not well know what to do, but eventually it lies down or falls down, and now the symptoms are the same whether after subcutaneous or intravenous injection. As the animal lies there the heart beats rapidly, strongly, and regularly; breathing, rapid and deep; perhaps there is myosis, and the retching continues.

When the dose is so large as to be speedily lethal, the respiratory movements become the longer the more laboured, while the heart's beat remains strong and regular; convulsions supervene, and are separated by intervals of complete voluntary motor paralysis. Finally, the respiration fails, a last convulsion with expulsion of the excreta, it may be, ensues, and death takes place, though the heart goes on beating for some time after the stoppage of the respiratory movements.

When the dose is not so rapidly lethal, and especially if administered subcutaneously, the course of the case may be subacute or chronic, and then the following symptoms are noted:—Violent vomiting and absolute inability to retain anything in the stomach, not even a little water or milk. Diarrhœa may be serous, but I have never seen it bloody; it may be persistent, or may recur from time to time only; tenesmus may be marked. Stomatitis accompanied by difficulty of chewing and swallowing, loss of appetite, great thirst, blackening of the teeth, softening of the gums, and the emission of a frightful odour from the mouth. There is almost constant eructation of ill-smelling gas from the stomach. The fæces in subacute and chronic cases contain nickel or cobalt, as the case may be, become nearly

black, and have a peculiar and characteristic odour. The urine of nickel cases remains of normal colour; but that of cobalt cases becomes of a rich brown colour, of a tint in proportion to the dose of cobalt, and persisting so long as cobalt remains to be excreted—that is, sometimes many days. Meanwhile, the animal gets emaciated if the case be chronic, and the heart's action somewhat weakens. The microscope shows no marked change of the red blood-corpuscles. The striking symptoms are those referable to the nervous system. They are in general very similar to those found in the case of rabbits. In one case the dog had convulsions decidedly epileptiform in character, a distinct tonus preceding the clonus; another had violent spasmodic strabismus. In a cobalt dog there was distinct dyspnoea from spasm of the glottis, and another had well-marked trismus. The reflex excitability may meanwhile be greatly exalted.

If recovery ensues, then the symptoms simply slowly abate; but if death ensues, the motor excitement becomes the longer the worse, the cramps are separated by intervals of paralysis, the breathing becomes embarrassed, and death ensues as in the more acute cases.

Post-mortem examination of different cases may disclose the blood of a very dark colour, and coagulated or not; heart with the auricles filled with blood, the ventricles pale, semi-contracted, or contracted; not in the mesocardium but in the endocardium and in the epicardium are small extravasations, mostly numerous; they are most numerous in the epicardium of the auricles, least numerous in that of the right ventricle; in the endocardium they are most numerous in the left ventricle and on the mitral valve. I have seen them extend through the whole thickness of a columna carnea or of a papillary muscle, but never down into the muscular substance of the heart's wall. Very frequently there are large decolorized clots in the auricles. I have rarely noted punctiform extravasations in the pleuræ, and never in the lung's substance. The mucosa of the stomach and intestine—particularly the small intestine and rectum—may be simply congested, or there may be extravasations into its substance. They are most constant and most marked in the stomach and around the pylorus; these may have become quite confluent, and may have become ulcers covered by a slough. In the stomach may

be masses of undigested food which have lain there for days and acquired a most offensive smell, and this doubtless accounts for the eructed foul gas. In the intestines mostly a bile-stained, never bloody, slimy fluid. In one case were infarcts of the spleen, and in two others conjunctivitis. Nervous system quite normal, though sometimes trivial congestion spots in the pia mater over the cerebral convolutions were noticed. In the case of one nickel cat the mucosa of the intestine was covered by a yellowish membrane about 1 mm. thick, and consisting of epithelium and mucus chiefly; the whole epithelium seemed to have become detached.

We thus see that the symptoms in connection with the alimentary tract are more prominent than in the case of rabbits. All along the tract there is an inflammation. The vomiting is an extremely prominent symptom, and may appear in a few minutes after the poison has been injected intravenously or subcutaneously, and its persistence and violence seems to exhaust the animal immensely. The thirst leads to the ingestion of much water, when that can be borne, and this increases the quantity of urine passed. I do not think that these metals have any specific diuretic properties as Azary (*loc. cit.*) seems to state. What may be the cause of these congestions and extravasations is not very clear; in the intestine we might assume an inflammation, but in the epi- and endocardium there is no evidence of this, and here of course the local action of an irritant is out of the question. As Schmiedeberg remarks, we have here, as in arsenious acid cases, conditions wonderfully like those found in Asiatic cholera and in poisonings by sepsin.

It is possible that these respiratory changes may be due to, first, an excitement, and then a paralysis of the centre in the medulla, for we shall see that another centre in the medulla is affected, viz., the vaso motor.

Harnack found these motor symptoms as well marked in lead poisoning of dogs, but not of rabbits, and ascribes them to excitement of brain centres higher than the medulla, from their peculiar constant jerking character, the non-affection of other centres in the medulla, and, lastly, from the fact that section of the spinal cord stops them. In arsenic and barium poisonings they are present, but are not marked, while in platinum and

acute iron poisonings they seem to be awaiting. In mercury poisoning v. Mehring found this motor excitement in cats expressed as movements like chorea minor in man.

### *I. Blood Pressure Experiments.*

These were carried out on rabbits, on cats, and on dogs, the cannula being in the carotid, and the metal, nickel or cobalt, solution injected intravenously.

Beginning from 10 to 15 seconds after the injection there is in all cases a remarkable fall of the arterial blood pressure, and while with the smaller more slowly injected doses the fall may be recovered from, with the larger and more rapidly injected doses the fall is continuous till death. The pulse rate is at first increased, but afterwards it is decreased. We shall see, however, that the pulse is not specifically affected.

It was necessary to assure myself by means of control experiments that these effects were not due merely to the injection of a saline solution. A quantity of solution of neutral sodic citrate, equal in bulk and in saline contents to the metal solution employed, and rendered just alkaline, was similarly injected. In some 10 seconds the pressure began to fall, the pulse to become slow and sometimes irregular; in about 10 seconds more the pulse began to quicken and the pressure to rise; and now the pressure remains higher than the normal for a minute or so, and gradually returns to the normal, as does the pulse. Now, since neutral sodic citrate differs from the metal double salt only in that the indifferent soda replaces the nickelous oxide, probably the fall of pressure in the experiments is exclusively due to the metallic oxide.

This fall of the arterial blood pressure may be due either to diminished force of the heart's action or to dilatation of blood-vessels.

That it is not due to the heart is certain, because, first, the muscle work experiments showed that striped muscle was not affected by the poisons; second, when the force of resistance is raised, either by abdominal compression or by stimulation of the spinal cord in the neck, the heart shows itself perfectly fit to cope with the increased resistance and to maintain the average blood pressure at a high level; third, the pulse remains practically

unaffected ; fourth, if the thorax be opened after death, the heart is seen to beat for several minutes, and the rhythm remains undisturbed, and if the fundus oculi be observed, the columns of blood in the vessels are found still unbroken. Section of the vagus does not influence this fall, whether performed before or after the metallic injection, nor does the administration of atropia.

The acceleration of the pulse coincides with the fall of the pressure, and is seen only when the vagi are intact, and consequently, according with Marey's law, the acceleration is probably simply due to the fall of pressure. As the case goes on the pulse becomes slower and slower, but this in all probability is to be ascribed simply to the defective nutrition of the organ, owing to the low blood pressure and the lowered temperature of the body.

In one solitary case of a dog (Expt. 65) there seemed to be a distinct slowing of the pulse ; the nervous system of the dog was severely affected, and evidently there was some vagal excitement.

That the fall is probably due entirely to dilatation of the blood-vessels is shown conclusively by the facts that abdominal compression raises the pressure very markedly, and that stimulation of the spinal cord in the neck raises it to a remarkable extent ; this latter shows that the efferent vaso motor nerve fibres, the endings in the smooth muscle, and the smooth muscle of the vascular walls remain unaffected, and that therefore the vascular dilatation is probably due to paralysis of the vaso-motor centre in the medulla. This conclusion is supported by the fact that complete suffocation of the animal raises the blood pressure less and less, and finally ceases to have the slightest influence in the way of raising the blood pressure, but rather causes a fall of it, probably due to insufficient respiration of the heart's substance. This loss of excitability of the vaso-motor centre takes place very early in the case, while yet stimulation of the cord in the neck brings about the remarkable rise in the blood pressure. Thus it cannot be argued that this paralysis of the vaso-motor centre is merely due to death of its tissues, seeing that the other tissues of the body for the most part retain their vitality. Therefore it is a specific action of the metal.

From the study of a nickel case it would seem as if the vaso-



motor centre, like the other motor centres, is first rendered more excitable and then gradually becomes paralysed, for successive suffocations of 20 seconds each, at intervals of about a quarter of an hour, showed the following rises of pressure in succession :— $10\frac{1}{2}$ , 36,  $40\frac{1}{2}$ , 26, 14, 6 mm. Hg., while the next suffocation for 20 seconds caused a fall of pressure of 1 mm. Hg., and so did suffocation during the succeeding 20 seconds.

Kebler found that stimulation of the peripheral end of the cut spinal cord raised the blood pressure in platinum poisoning less and less as the case progressed, and infers from this that platinum exerts a curara-like action on the endings of the nerves in the muscle of the vascular wall. In my experiments I also found this lessening of the rise, but in a case where the pressure had fallen so low as only 2 mm. Hg., and the dog had been curarised, and had been having its blood-pressure noted during a period of some five hours, the cervical stimulation was still able to raise the pressure to 55 mm. Hg. During this time also the dog had frequently been suffocated, and its spinal cord had frequently been stimulated, so that the vitality of the tissues was reduced to a minimum, and therefore heart and blood-vessels can no longer answer to the stimulus as they did before.

Azary says that the fall is due to a purely local action on the heart's muscle and ganglia; with that, of course, I cannot agree.

Harnack found that lead had no influence on the vaso motor or on the vascular system. Meyer and Williams note the fact that in acute iron poisoning of cats suffocation does not raise the pressure when it has fallen very low, and Unterberger found that in arsenious acid cases neither direct nor indirect stimulation of the vaso motor centre sufficed to raise the pressure; and finding that stimulation of the peripheral cut end of the great splanchnic no longer contracted the vessels of the corresponding area, while stimulation of the sympathetic always contracted the aural vessels even after death, he suggests the possibility of a local vaso motor paralysis in the abdomen. He states that there is some cardiac paresis. Von Mehring found also for mercury some cardiac paralysis and peripheral vascular paralysis.

Boehm and Mickwitz found that, even after section of the spinal cord, small doses of barium salt injected intravenously

raised the pressure very remarkably, though it afterwards fell to the normal, or, with the smaller lethal doses, to the abscissa, and death, while the large lethal doses simply caused a continuous fall till death. They exclude the heart, and leave it undecided whether the rise is due to a direct action on the vascular muscle, or indirectly through the nerve-endings.

Nickel and cobalt seem to resemble arsenious acid most in their action on the vascular system, as indeed they also do in their action on the alimentary tract, and to a certain extent on the nervous system. With regard to the other metals, I am convinced that much must yet be done in the way of experimentally localising their action.

#### *K. Absorption and Excretion of the Salts of Nickel and Cobalt.*

Frogs painted with the solution gradually develop the symptoms of the poisoning. From the stomach absorption does take place, because the metal may be detected in the urine and in the fæces, and even death may be caused in this way, but with difficulty. For instance, a rabbit had introduced into its stomach at intervals of six days successive doses of 100, 200, and 400 mgr. of NiO pro kilo. animal, and showed no symptoms till after the last dose, when it died in eight hours, with symptoms of adynamia merely; and a dog had 500 mgr. NiO pro kilo. dog in small doses within fifteen days, and never showed anything but several slight vomitings. I have never noticed any indication of local irritation at the point of injection—all the solution being rapidly and entirely absorbed and diffused.

Excretion of the metals is accomplished chiefly by the urine, in which they may be detected for many days after the administration of a single dose. For its detection and isolation I evaporated the urine, incinerated the residue, extracted the ash with hydrochloric acid, and from this extract threw down the black metallic sulphide with the sulphuretted hydrogen stream. In the bile the metals were similarly detected; also in the fæces large quantities were found, but how much was due to the bile and how much to the intestinal secretion I did not determine. That some of the metal is got rid of by the intestinal secretion is evident from the fact that high up in the cæcum of rabbits killed very soon after the subcutaneous or intravenous injection

of the metal the metal could easily be detected. This was so soon after the administration of the poison that no bile could by that time have penetrated so high up the cæcum, and care was taken that there should be no laceration of tissue in any way, nor admixture of blood.

The nickel urine does not alter its colour, but the cobalt urine is of a rich dark brown colour, the depth of the colour being in exact proportion to the quantity of cobalt present. If such a urine be allowed to decompose, it deposits a crop of ammonio-magnesian crystals of the ordinary form, but of a most beautiful violet or purple colour, various tints being found together. This I have found an excellent test for the cobalt.

I have not yet determined the nature of this brown cobalt compound, for such it is, no trace of blood-pigments or their decomposition products being present in the urine. Such brown urine, treated with basic acetate of lead, yields a brown precipitate and a filtrate which still contains plenty of cobalt, so that the metal is excreted in more than one form. The brown lead precipitate washed with hot water, and then extracted with carbonate of ammonia, yields a solution of the same colour as the original urine.

#### L. Lethal Doses of Nickel Oxide.

The subcutaneous lethal dose is given as nickelous oxide for a kilogramme weight of animal, and is probably as follows:—

Frogs, . . . .	·080	Cats, . . . .	·010
Pigeons, . . . .	·060	Rabbits, . . . .	·009
Guinea pigs, . . . .	·030	Dogs, . . . .	·007
Rats, . . . .	·025		

The number of cobalt experiments was not so great as in the case of nickel; but from the records of twenty-one experiments cobalt seems to be only about two-thirds as deadly as nickel.

The following are the records of several experiments selected from the hundred that were included in my Thesis, and the numbers refer to the Thesis:—

*Experiment 6.*—A male *Rana temporaria*, weight 37 grammes, and a gravid female *R. temp.*, weight 80 grammes, poisoned each with ·1 NiO per kilo. frog.

*January 4th.*—Solution injected into dorsal lymph-sac. Both are paresed, show twitchings of the toes, and incoordinated movements particularly of hind limbs. Both can walk, but not hop; nor turn round from the dorsal position; and both appear stupefied. Female is more gravely affected than the male.

*5th and 6th.*—Same condition.

*7th.*—Can now turn round.

*8th and 9th.*—Same condition.

*10th.*—Both walk well enough, but do not yet hop; will lie on their back, but turn round on being excited.

*11th to 14th.*—Gradual recovery; now almost well.

*15th.*—Male but slightly affected; female weak and apparently dying.

*16th.*—Same condition.

*17th.*—Male pretty normal; female still weaker; reflexes almost gone.

*18th.*—Male still has symptoms; female dead; gastric mucosa showed doubtful congestions.

*19th and 20th.*—Little to be noticed except some drawing up of the legs.

*21st.*—Drawing up of legs is more pronounced than it was yesterday.

*February 1st.*—Still some drawing up of the legs.

*12th.*—Now it is seemingly normal.

*Experiment 51.*—*Rabbit* ♂.—Weight, 1800·0; poisoned with ·008 NiO pro kilo. rabbit injected into the blood.

*February 10th, 11.40.*—Injection practised, slight cramp-like contractions accompany it; pulse 152.

6.0.—There has been further diarrhoea, but no other symptom; pulse regular and good, 156.

*11th, Morning.*—More diarrhoea in the night; heart rapid, not easily counted.

*11th, Evening.*—Pulse good, 112; no further diarrhoea and no spasms. (It has a sneezing, but we do not know if it is something new; nothing was previously remarked.)

*12th.*—Eats very little; heart pretty good; pulse 112; sneezing still persists.

*13th.*—Has eaten nothing; heart fairly strong; pulse pretty regular, 88; no spasms; sneezing pretty violent.

*14th.*—Has not eaten during past 24 hours; pulse regular, 108.

*15th.*—Difficult to say whether pulse is 240 or 120.

*16th.*—Feeds well, and appears perfectly normal.

*17th.*—Same condition.

*18th.*—P. 136; diarrhoea; but the animal is tolerably well notwithstanding.

*19th and 20th.*—No more diarrhoea, but is enormously excitable; jumps about when one wants to touch it, and has a sort of shiver when one gets hold of it.

*21st.*—P. 136; nothing noteworthy.

22nd.—P. 144 ; apex beat good. Again frightful diarrhoea. Faeces thin, fluid, or gruel-like ; brown, but no blood in them. Again the animal is in the excitable condition before noticed.

23rd.—P. about 148 ; violent diarrhoea in the night ; eats tolerably well ; heart somewhat irregular.

24th.—P. 124, regular and good ; feeds well ; violent diarrhoea, which has nothing to do with the feeding, which is quite dry, and has no such effects on other rabbits ; weight 1530 grms.

25th.—Status *idem*.

26th.—P. 140 ; still diarrhoea ; fundus oculi certainly paler.

27th.—Status *idem*.

28th.—P. 124, good ; diarrhoea in the night.

March 1st.—P. 120, good ; feeding well.

2nd and 3rd.—P. 120 ; diarrhoea not so marked.

4th.—Very severe diarrhoea during the night ; had leaped out of the cage when the servant came in the morning ; sprang round the room in an extremely excited condition, so that he could scarcely catch it. In the evening respiration 78 ; pulse 280, good ; and it was still excited as before.

5th and 6th.—P. up to 300 on repeated countings ; diarrhoea persists.

7th.—Diarrhoea is now less.

12th.—Diarrhoea has continued more or less always. To-day is end of session. I wanted to utilise the animal for a blood-pressure experiment, but it died before the experiment was well begun. The only thing pathological was a few old extravasation spots in the gastric mucosa.

*Experiment 52.—Rabbit ♂.*—Weight, 1685·0. With 0·01 NiO pro kilo. rabbit injected into the blood.

January 3rd, 11.0.—P. 140, R. 100. Injection into the crural vein. After the injection the pulse and respiration are greatly quickened ; no diarrhoea, but passage of urine, and cramp-like movements, though not very marked.

11.15.—Let loose from the board ; it is not paralysed, but seems stupefied, so that it lies on its side, with the pupils a little contracted.

11.20.—Lying on its side, rises on being molested ; its hind legs are the most affected, they are both to one side, while the fore legs are thrown outwards, one on each side. Pulse still quick ; breathing quiet, and so it remains until

1.0.—P. 132, R. 88.

3.0.—P. 124, R. 140. Appears quite well, no more lameness nor pupillar contraction.

6.0.—P. 132, R. 140. Status *idem*.

4th, 9.0.—P. 132, R. 92. Apparently normal.

6.0.—P. 92, R. 100.

5th, 9.0.—P. 112, R. 72. Jerkings all over the body, has a sort of rigor, and cowers when one touches it. Pupils normal.

7.0.—P. 96, R. 120. The movements are still present, but they are only slightly marked, and are passing.

6th, 10.0.—P. 100, R. 56. Jerkings always here yet.

6.0.—P. 132, R. 12. It has not been feeding properly to-day; the jerkings are less marked than in the morning, but are still present, and when it was first touched it showed peculiar manège movements.

7th.—Pulse very rapid; it has fed during the night. Shiverings all over the body.

8th, 5.45.—Hitherto it has been in *statu quo*, but now it has a violent convulsion, whereafter it lies on its side as if it were dead.

5.50.—Another little shiver, then it recovers and sits still quietly. P. 120, R. 72. Apex beat irregular in strength, and shiverings are easily called forth by touching it. It is so weak that it falls off its feet, so to speak, the fore legs going outwards as usual. In a little while it has a perfectly complete general contraction, tetanus lasting a minute. The ear vessels during this time enormously enlarged, as were also the retinal vessels, seen by the ophthalmoscope. Several little contractions follow.

6.45.—P. 220, R. 120. Pulse irregular in time. The ears have remained untouched for a whole hour, and yet the vascular dilation is as great as ever.

6.56.—Another convulsion, and passage of urine.

7.0.—Death. The vessels became quite flaccid and pale, then the respiration ceased. With the ophthalmoscope the breaking of the retinal blood columns was watched, and on this ensuing the chest was immediately opened, and the heart was found beating—without any exposure to the air.

*Post-mortem Examination.*—Two groups of little ulcers in process of healing covered by a scab at the beginning of the pylorus; in the intestine nothing to be seen; contents copious. In the bladder ten extravasation spots. Heart normal, not contracted nor dilated. Everything else normal.

*Experiment 58.*—Rabbit ♂.—February 10th.—Weight, 1810.0. With .012 NiO per kilo. rabbit, subcutaneously. P. 208, regular, just before injection.

10.0.—Injection. After the injection it showed little, except that it is quieter and less brisk than it was; it was extremely active beside the females, now no longer so.

6.0.—At 6.0, when the pulse = 152.

11th, 10.0.—Pulse very much accelerated, and not to be counted. Tries to feed, but cannot. Sits as if stupefied. When it is lifted up by the ears there appears at once in the legs, particularly the fore legs, a regular continued tremor, which gives one quite the idea of the ankle clonus of lateral sclerosis in man; it is on both sides equally, is stopped by the very slightest pressure made on the limb, but continues in the opposite one unaltered. When it is now set down on the ground the head and ears move for a while, just as in other such nickel cases, giving one the idea of paralysis agitans of man.

4.0.—P. 120, R. 88. Mostly paralysed. Cries from time to time spontaneously; breathing difficult, and evidently with an effort. On pinching the legs the reflexes are exceedingly violent, but meanwhile

it simply lies there as if it had lost all power of voluntarily moving. The blood pressure was now sought to be ascertained. From the very first the pressure was at a very low level, and it sank still lower for some time. The animal died with convulsions, evidently due merely to stoppage of the respiration. Shortly before death the vessels of the fundus oculi were not dilated.

*Post-mortem.*—Nothing pathological.

*Experiment 70.*—*Cat.*—Weight, 2450 gram. With 10 mgrm. NiO pro kilo. cat. Subcutaneously.

*August 11th, 1.0.*—Injection: cat does not seem very well even now, i.e., before the injection.

4.45.—Weak, dejected, and dull; has vomited, and vomits still; heart normal.

5.30.—Was seen by laboratory man; was ill, crying as if in pain, vomiting severely, convulsions at intervals, hind legs shaken violently, and it is bent backwards; after a fit it lay as if dead, but when molested could rise, but fell immediately.

5.45.—The man left it apparently dying.

8.0.—I found it dead, still warm, on its side; no salivation; no evidence of diarrhoea; muscles still directly excitable; heart normal, auricles distended by dark fluid blood, ventricle contracted, no dilatation of abdominal vessels to speak of. Intestinal mucosa covered by a greenish-yellow layer 1 mm. thick, which, examined microscopically, was ascertained to be entirely composed of epithelial cells shed from the villi; the layer separated quite easily from the subjacent mucosa, which was congested. Bladder empty and contracted.

*Experiment 61.*—*Dog, young ♂.*—Weight, 6800.0. With .005 per kilo. dog into blood.

*February 9th.*—P. 110, before operation.

4.0.—Injection: not followed by cramps, defæcation, nor urination.

6.0.—Appears quite recovered; eats when flesh is offered him.

10th, *Morning.*—P. 140. Does not eat just so eagerly as yesterday.

„ *Evening.*—Is somewhat out of sorts.

11th, *Morning.*—P. 152, good and regular. Is cheerful enough, but makes no longer any effort to jump out of the cage, and though he takes the bits of flesh into his mouth he lets them fall again. No fæces since the operation. Did not eat his supper last evening.

*Evening.*—P. 148. Has not eaten anything during the day; this evening drank an enormous quantity of water, but immediately vomited it again, and with it he has brought up the flesh which he ate yesterday, quite undigested and stinking. Here, as in almost all other such cases, great quantities of stinking gases are continually being brought up from the stomach.

12th.—P. 144. Ate only half his supper last night, the other half this morning, but would not eat any further flesh offered to him. Great thirst, and much water drank, but no further vomiting. Still the eructations of stinking gas.

13th.—P. 168. Pulse very good. Only a part of the food eaten, but it was vomited again. Looking somewhat miserable.

14th.—P. 172, good. Eaten all his supper of last evening and retained it.

15th.—P. 160, pretty good and regular. Thirst, but eaten no flesh. A slight indication of conjunctivitis.

16th.—Status *idem*.

17th.—P. 200. Eyes better. Has taken his food, but will have no more from the hand.

18th.—P. 72. Great thirst. Has taken all his food. Fæces fluid and of a very dark colour, as in all such nickel dogs.

19th.—Status *idem*.

20th.—Do. do.

21st.—Evidently normal.

22nd.—Do. do.

23rd.—Do. do. A good deal of nickel still in the fæces.

24th.—Do. do. Hard fæces, and at intervals of two days or so.

25th.—Do. do.

26th.—Has some diarrhœa, but is cheerful and well-looking.

27th and 28th.—Nothing noteworthy. He may be considered normal, only the stomach is not quite well; still he has the peculiar nickel-smell, and there is some conjunctivitis. Now he is used for a blood-pressure experiment, after which post-mortem showed numerous extravasations in the gastric and duodenal mucosa; otherwise nothing.

*Experiment 65.*—Dog, old ♂.—Weight, 9500·0. With ·008 NiO subcutaneously.

*January 8th.*—P. 120, R. 18; before injection.

3.40.—Injection practised.

4.10.—Urine passed, also thin fæces; appears restless.

4.40.—Lies down as if exhausted.

5.0.—Still is lying, and does not rise when one goes up to him; looks dull, and seems pretty ill.

5.30.—P. 124, R. 20. In the same condition; pupils normal.

7.0.—Is somewhat recovered.

6th, 10.0.—P. 180, R. 32. Lies very quietly, and looks really ill.

6.0.—P. 160, R. 24. Lies still in the same quiet feeble-looking fashion; he eats only three-fourths of his accustomed fare, and seems uneasy after that; he wants then to lie down again, yet appears not rightly to know how he should lie, and falls down rather than lies down, then remains quietly lying, growls or snarls from time to time.

7th, 1.0.—P. 132, R. 28. Vomited his whole breakfast this morning; vomited matter of fearful smell; lies completely on his side; growls no longer; sometimes moves in a shivering manner his legs; pupils contracted. When molested, he rises with difficulty, and immediately falls, rather than lies down again. Seems weaker in the hind than in the fore legs. Has drunk much water, but almost immediately vomited it again, and with it brought up some undigested pieces of meat.

4.0.—Somewhat better.

5.30.—P. 132, P. 22. Quite still, looks somewhat cheerier. Has passed no fæces since above noted. Urine contains large quantities of nickel.



8th, 10.0.—P. 120, R. 30. Somewhat better; again vomited some undigested flesh of the same smell. Did not eat his supper last night, and it was found this morning wholly untouched, so that the vomited flesh had remained in the stomach—it may be twenty-four hours—since yesterday morning. Seems quite intelligent. Drinks an enormous amount of water. From his bent-up mode of standing, and from his wincing on pressing his abdomen, he seems to have pain in that region. His whole body shivers remarkably, and in his right eye occasionally are tremulous contractions of certain of the external muscles, and so strabismus. The heart is feeble, and from this and from the marked contraction in the muscles of the chest, it is difficult to observe and count the heart-beat. Owing to contractions of the neck muscles, the head has quite the movements of paralysis agitans in man.

1.0.—P. 120, R. 22.

7.0.—P. 112, R. 26. Lying quite quietly; but appears better a little.

9th.—P. 124, R. 20. Better to-day; eaten three pieces of flesh, and retained them.

10th, 10.0.—P. 120, R. 14. Brisk; drinks a great deal of water. Heart-beat regular and strong.

4.0.—P. 36, R. 14. Lies bent up, breathes quite quietly; appears weary. Heart, as well as slow, is very irregular. Breathing is thoracico-abdominal in type.

7.0.—P. 43, R. 14. Lies as before; pulse now regular. Still as thirsty. Has not eaten much, only a little flesh and milk; but that he has retained.

11th, 10.0.—P. 51, R. 14. Heart irregular, though not so much so as it was yesterday. Eaten all his food. Passed fæces now for the first time since before noted.

1.0.—P. 42, R. 13. Heart quite regular in time and strength. Lies quietly.

5.0.—P. 44, R. 13. Heart somewhat irregular; but strong enough. Appears sleepier than before.

12th.—P. 102, R. 15. Quiet; eaten all his food, nothing vomited. Looks normal.

13th.—P. 120, R. 20. Heart quite regular. Left some of his food over.

14th.—P. 120, R. 14. Apparently normal.

15th.—P. 54, R. 12. Eaten little or nothing. Heart strong, but somewhat irregular.

16th.—P. 120, R. 16. Has eaten nothing. Vomited some of yesterday's food still; is still very thirsty. Heart regular, but somewhat weak as before.

17th.—P. 140, R. 18. Has eaten a good deal of flesh, and drunk much water. Heart regular. Drinks and eats with difficulty, seeming as if he had not power in the jaw muscles, and then swallowing is evidently only accomplished with an effort. Urine, of twenty-four hours' collecting, showed traces of nickel.

18th.—P. 156, R. 16. Pretty brisk; has eaten all its food; great thirst; the same difficulty in eating and swallowing. Heart regular.

19th and 20th.—P. 156, R. 20. Heart regular. Is lively; has eaten all his food; looks now clean and well. Seems quite recovered.

21st.—P. 124, R. 17. Heart regular and good. Lively, and to-day, for the first time, he wants to spring out of the cage; but he has vomited all that he ate yesterday evening and this morning, and when fresh flesh was offered him, he would not touch it, so that there was a distinct disinclination to eat. The vomited was a hard dry mass of undigested food.

22nd.—P. 120, R. 20. Eaten everything. Shows nothing abnormal.

23rd, 10.0.—P. 12, R. 18. Considerable diarrhoea; thin brown fæces.

3.0.—Has repeated convulsive attacks. When one comes, there is first a short stage, where the character is markedly tonic, and general all over the body, so that the neck and trunk are bent backwards, and the limbs stretched straight out from the body; then succeeds a most violent clonic stage, in which the whole body partakes, and where the movement is so violent that he is dashed about the cage from side to side. They come on very quickly, disappear more slowly, but still not just to say slowly. They succeed each other with growing frequency. Heart not observed, since all assured that to open the cage and handle him was decidedly unsafe.

4.0.—Found dead.

*Post-mortem Examination.*—Emaciated somewhat, but still much subcutaneous fat. Muscles dry. No trace of peritonitis. Three pieces of undigested flesh in the stomach. The gastric mucosa shows only a few small extravasation spots towards the pylorus, the mucosa is gall-stained and yellow, and the fluid contents of stomach are similarly yellow. In the whole intestine are no fæcal masses, only a yellowish-brown fluid, and this in the rectum becomes blackish. Throughout the whole intestinal mucosa were numerous reddish extravasation spots, especially in the duodenum and ileum, and in the rectum were many quite fresh red extravasations. Gall bladder full. Pancreas small, soft, and congested. Kidneys normal. Urinary bladder strongly contracted. The pleuræ and pericardium free from spots. Heart not contracted, nor yet flaccid, moderately full of fluid, dark, cherry, or violet coloured blood. Beautiful hæmorrhages under the serosa of the mitral valve. Especially in the auricles were large, white coagula, completely colourless, old. Nothing in nervous system.

*Experiment 88.—Rabbit, Male.*—Weight, 2110.0 grms.; with 0.020 CoO injected into the blood.

*February 26th.*—P. 162 before injection.

11.0.—Injection; after the operation pulse is 128; it lies down as if exhausted; pupils not contracted; respiration normal.

12.0.—P. 136, good; respiration normal; sits close to the floor as if weary or afraid, but rises quite well when molested.

12.15.—Suddenly it falls down and lies on side; pulse 160, somewhat weak; pupils contracted; breathing slower and with difficulty.

12.20.—Pupils dilate; on trying to rise it cannot; the respiration is becoming slower and with greater difficulty, and the pulse becomes

slower; convulsion with opisthotonos appears; this now subsides, and then the breathing is with great difficulty; urine is passed, and it dies apparently from respiratory paralysis, for after the respiration has ceased the heart is felt to beat for some time; after that it cannot be felt; it is seen to beat on opening the chest, and the blood columns of the retina do not break for long after the cessation of respiration.

*Post-mortem.*—A few punctiform extravasations in the pleura pulmonalis of each side; also in the gastric and intestinal mucosa. No firm faecal masses in the intestines. Two endocardial extravasations of small size on papillary muscles, but meso-cardium quite normal.

*Experiment 95.—Dog, not young, Male.*—Weight, 5400·0; with 0·050 CoO into blood.

*February 21st.*—P. 82.

12.30.—Injection practised.

3.0.—P. 80; good, but not so regular as normally; no faeces passed; has vomited much frothy, tough, mucous matter; pretty brisk.

5.45.—Lies on his side; respiration difficult; from time to time spasms of clonic character and of single members; the faecal muscles very violently contracted, and so the teeth well shown. The respiration becomes more and more difficult, and is accompanied by a sound as if there were spasms of the glottis. During the spasms the heart is not to be felt, and only at

6.10.—When the animal is dying and the jerkings have ceased is the heart to be felt. Immediately after death the chest wall is opened, but pericardium is left intact; the heart beat quite well for 6–7 minutes longer.

*Post-mortem Examination.*—Twenty-four hours after death: rigor mortis; blood is dark and coagulated; pupils normal; gastric contents clear, fluid, and brownish coloured; intestinal contents very little, and of same nature as in the stomach; urinary bladder contains a few c.c. of dark brown urine; great oedema of the gastric and intestinal mucosa; towards the pylorus congestions and extravasations increase in number, and are most in duodenum and rectum; lungs, pleura, pericardium, liver, spleen, and pancreas all normal; some endocardial extravasations, none elsewhere in heart; veins of dura mater are greatly overfilled; brain is greatly congested, and has numerous smaller and larger spots that are doubtful extravasations; few of these are on the cerebellum, and none are in the white substance.

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A "KERATO-THYRO-HYOID" MUSCLE AS A VARIATION IN HUMAN ANATOMY. By S. G. SHATTOCK.

THIS muscle, which is well known in comparative anatomy, has not, so far as I can ascertain, been hitherto found, or at least described in man. It is noticed neither by Wood<sup>1</sup> nor Macalister.<sup>2</sup> The muscle, in comparative anatomy, is known as the kerato-hyoid; but as the same name has been applied by Gruber to a fasciculus sometimes found passing from the superior cornu of the thyroid cartilage to the greater cornu of the hyoid bone, it may be as well, perhaps, to make it clearly distinguishable by naming it, as above, from the parts between which it passes (kerato-hyal and thyro-hyal). Chauveau<sup>3</sup> describes the kerato-hyoideus in the horse as "a very small fasciculus attached on one side to the posterior border of the styloid cornu and inferior extremity of the styloid bone, and on the other to the superior border of the thyroid cornu;" he afterwards notices its larger development in the Carnivora. An exact description of the human muscle, in the case in which I observed it, may suffice without the requirement of a figure.

The muscle arises in a fan-like way for about one centimeter from the middle of the outer surface of the greater cornu of the hyoid bone, in a line with the middle constrictor of the pharynx, the origin of which from the cornu ceases a short way in front of the posterior limit of the muscle which here overlaps it.

The tendon of the muscle is inserted into the posterior margin of the lesser cornu in close relationship with those fibres of the middle constrictor which arise from the lesser cornu; some of the fibres of the middle constrictor, indeed, take origin from its tendon of insertion.

Its obvious action is to depress the lesser cornu, or approximate the lesser and greater cornua, should a movable articulation exist between the greater cornu and the body of the hyoid

<sup>1</sup> *Proc. Royal Society London.*

<sup>2</sup> "Muscular Anomalies in Human Anatomy," *Trans. Royal Irish Academy.*

<sup>3</sup> *Anatomy of the Domesticated Animals.*

bone (not the case in the specimen); and it would of course antagonise, when existing with it, the muscle referred to by Chauveau as the transversalis hyoidei—a short riband of parallel muscular fibres which unites the superior extremities of the styloid cornua and approximates them.

There existed, moreover, in this specimen, another proper or intrinsic muscle of the hyoid bone. This, which may be named "basi-thyro-hyoid," arose (or was inserted) in front of the preceding, from the greater cornu of the hyoid bone, and terminated in a tendon attached to the body beneath the articulation of the lesser cornu; the muscle was situated under cover of the basio-glossus, and lay, of course, on the bone throughout its length.

Professor Thane has met once with a muscle corresponding in its essential attachments to this, though differing somewhat in detail; it arose from the outer and lower part of the free extremity of the greater cornu, and was inserted by a slender tendon into the body of the hyoid bone below the origin of the basio-glossus,—it existed on the right side only: it differs, therefore, from that described above, in its being superficial to, instead of beneath, the basio-glossus, and in its greater length.

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### Rebiew.

CESALPINO AND HARVEY. *The Harveian Oration delivered at the Royal College of Physicians of London, June 24, 1882.*  
By GEORGE JOHNSON, M.D., F.R.C.P., F.R.S.

WHEN in Rome last spring, conversing with Dr. Pantaleoni and others, I found that the feeling in favour of Cesalpino's claims to be regarded as a discoverer of the circulation of the blood, in preference to those of Harvey, are very decided, and that they attribute to English prejudice the honour which in this country is ceded to Harvey. I was glad, therefore, to find that Dr. George Johnson has devoted his recent *Harveian Oration* to a careful exposition of the views of Cesalpino, deduced, partly from Cesalpino's writings, and partly from a work by Ceralдини, a professor of physiology in Geneva, "wherein," according to an oration by Professor Maggiorani, made on the occasion of the unveiling of a monument to Cesalpino at Rome in 1877, "that which heretofore had been only more or less a belief as to Cesalpino's discovery, has become, by means of new arguments, a scientific demonstration." Ceralдини's statement with regard to Harvey Dr. Johnson gives as

follows :—"That during the four years, from 1598 to 1602, which Harvey spent as student at Padua, he must have become acquainted with Cesalpino's writings, some of which had been published about thirty years before; that in these writings Harvey must have seen that the true doctrine of the circulation of the blood was clearly set forth and demonstrated; that Harvey designedly delayed the publication of his work, *De Motu Cordis et Sanguinis*, until 1628, twenty-five years after the death of Cesalpino, and nine years after the death of Fabricius, when his adversaries could adduce no proof that his affected ignorance of the discovery of Cesalpino was a mere pretence." Dr. Johnson then proceeds to show "that, however diligent may have been Harvey's study of Cesalpino's writings, he could never have obtained from them that which is not to be found therein, viz., a knowledge of the circulation of the blood; and that those who pretend to find in these writings the true doctrine of the circulation, endeavour to establish their position by giving to some chance expressions a meaning which the context shows could never have been in the mind of the author."

The following inscription and paragraphs are copied from a memorandum card printed on the occasion of placing a bust of Cesalpino in a new hall built in the University of Pisa in 1878. For the opportunity of seeing this card I am indebted to Dr. Johnson :—

Andreas Cæsalpino, Domo Aretico  
Archiatro eximio  
Solertissimo Naturæ Investigatori  
Quod In Generali Sanguinis Circulatione  
Agnoscenta et Demonstranda  
Cæteros Antecesserit  
Plantas Nondum in Species Tributas  
Primas Ordinandas Susceperit  
Rerum Plurinarum Impeditam Intelligentiam  
Explicuerit  
Universam Morborum Doctrinam  
Magno cum Plausu in Hoc Archigymnasio Tradiderit  
Sodales Medici  
Et x viri Archigymnasio Moderando  
Honoris et Memoræ Causa  
III. Prid. Calend. Octol. MDCCLXXVI.

"FUGIT ENIM SANGUIS AD COR TANQUAM AD SUUM PRINCIPIUM non ad hepar aut cerebrum, quod si cor principium est sanguinis, venarum quoque et arteriarum principium esse necesse est, vasa enim hæc sanguini sunt destinata."—Cesalpino's *Quæst-Peripatet*, lv. quæst. iii.

"MOTUS igitur continuus a corde in omnes corporis partes agitur, quia continua est spiritus generatio, qui sua amplificatione diffundi celerrime in omnes partes aptus est; simul autem alimentum nutritivum fert, et auctivum ex venis elicit per osculum communicans quam Græci anastomosin vocant."—*Ibid.*, lv. quæst. iv.

"Idcirco pulmo per venam arteriis similem ex dextro cordis ventri-

culo fervidum hauriens sanguinem, eumque per anastomosin arteriæ venali reddens, quæ in sinistrum cordis ventriculum tendit, transmissio interim aere frigido per asperæ arteriæ canales, qui juxta arteriam venalem protenduntur, non tantam oculis communicantes, ut putavit Galenus, solo tactu temperat. HUIUS SANGUINIS CIRCULATIONI ex dextro cordis ventriculo per pulmones in sinistrum ejusdem ventriculum optime respondent eo quæ ex dissectione apparent.”—*Ibid.*

“Sed illud speculatione dignum videtur, propter quid vinculo intumescunt venæ ultra locum apprehensum non citra; quod experimento sciunt qui venam secant; vinculum enim adhibent citra locum sectionis, non ultra, quia TUMENT VENÆ ULTRA VINCULUM NON CITRA. Debuisset autem opposito modo contingere, si motus sanguinis et spiritus a visceribus fit in totum corpus; intercepto enim meatu, non ultra datur progressus; tumor igitur venarum ultra vinculum debuisset fieri.”—*Quæst. Med.*, l. ii. quæst. xvii.

“Sciendum est cordis meatus ita a natura paratos esse, ut ex vena cava intromissio fiat in cordis ventriculum dextrum, unde patet exitus in pulmonem, ex pulmone præterea alium ingressum esse in cordis ventriculum sinistrum ex quo tandem patet exitus in arteriam aortam, membranis quibusdam ad ostia vasorum oppositis ut impediatur retrocessum; sic enim PERPETUUS QUIDAM MOTUS EST EX VENA CAVA PER COR ET PULMONES IN ARTERIAM AORTAM, ut in Questionibus peripateticis explicavimus.”—*Ibid.*

“Nam in animalibus videmus alimentum PER VENAS DUCI AD COR TANQUAM AD OFFICIINAM CALORIS INSITI, ET ADAPTA IBI ULTIMA PERFECTIO, PER ARTERIAS IN UNIVERSUM CORPUS DISTRIBUI agente spiritu, qui ex eodem alimento in corde gignitur.”—*De Plant.*, l. i. c. ii.

From the above, which may be supposed to give no unfavourable statement of Cesalpino's views, and from additional passages quoted from his works by Dr. Johnson, we learn that he understood the passage of the blood from the vena cava into the right auricle, through the pulmonary arteries and veins to the left auricle, and through the left auricle and ventricle to the aorta; that he understood, in short, the course of the blood through the heart, the disposition and action of the pulmonary and aortic valves, and the pulmonary circulation, except that he considered the blood to be transmitted from the pulmonary arteries to the pulmonary veins by “anastomosis.” He thought that the blood, full of heat and spirit, generated in the heart, was tempered as it passed through the lungs by the proximity of the air in the bronchial tubes. With regard to the systemic circulation he recognised that some of the blood passed from the aorta through anastomosis to the vena cava and back to the heart. He thought that the arteries carried *nutritive* blood to the tissues and derived from the veins, through anastomosis, *auctive* blood, which was also sent to the tissues. The blood thus sent to the tissues did not, it may be inferred, according to his view, traverse them and reach the veins, but became coagulated or fixed in the tissues. The transfer of blood from the arteries through anastomosis was not therefore continuous. Indeed, he thought the blood chiefly coursed through the arteries, and, to some extent, through the veins to the tissues during the day, and through the veins to the heart



during sleep, there being something of a flux and reflux in both arteries and veins. He recognised that the fact of the swelling of the veins beyond a ligature proved that the blood did not flow from the organs through the veins to the body. He had no idea of a capillary circulation, or, indeed, apparently of any transfer of blood through the tissues. The term "*capillamenta*" which he uses, meaning, not capillaries in the modern sense of the term, but the fine hair-like nerves into which he considered that a certain amount of the blood passes without apparently passing through them. It may be said then that he had a generally correct idea of the course of the blood in the heart, and in the large vessels of the lungs and the system, though he thought that there was sometimes a reflux of blood in the arteries and especially in the veins, and that the reflux current in the veins supplied auctive blood to the arteries for the tissues. Dr. Johnson, however, shows that in his ideas he was little or not at all in advance of Galen and his other predecessors, and indeed in the statement in one place made by him that some of the blood passes directly from the right ventricle to the left, through the pores of the septum, he was behind Servetus and Columbus, who had disposed of that view some years before he wrote. The passage of blood through the tissues, both in the lungs and the system, seems to have been unknown to him, and he had no idea of that which was Harvey's great discovery, the motor force of the heart. His notion was that the heat and spirit generated in the heart were, by expansion, the motor force, and that the thickness of the coats of the pulmonary artery and the aorta was for the purpose of preventing the escape of this spirit at the part where its expansive force was greatest.

While, therefore, we may admit to Cesalpino and other Italian anatomists, to Galen and Servetus, a greater amount of knowledge respecting the circulation than is sometimes accorded to them in this country, and perceive that in their general view of the course of the blood—that is, of a circulation through the heart, large arteries, and veins of the lungs and of the system—they were correct; it is evident that they did not understand that very important part of the circulation, which consists in the passage of the blood through the tissues—the manner, that is, of the passage of the blood from the arteries to the veins, or of a continuous flow in any one direction. A clear, concise, correct view on these points, respecting which they were in great confusion, and more particularly the knowledge of the heart's action and of the force by which the circulation is effected, respecting which they were quite ignorant, were left for Harvey to work out. This he did in the careful, masterly manner that has made his name so great.

G. M. HUMPHRY.

# Journal of Anatomy and Physiology.

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**A METHOD FOR THE ESTIMATION OF UREA IN THE BLOOD.** (Containing also a Method for its Estimation in Muscle, and a Series of Experiments as to the Variations in its Amount in the Body within Physiological Limits.) By **JOHN BERRY HAYCRAFT, M.B., F.R.S.E.,** *Professor of Physiology in Mason College, and Lecturer on Physiology in Queen's College, Birmingham.*

## PART I.

**THE** estimation of a quantity of urea in a pure watery solution is not difficult; nothing, indeed, can well be easier. Many methods may be employed, which are both exact and easy of application; such are those of Liebig, of Heintz and Ragsby, of Bunsen, of Hüfner, and of Davy. Before estimating the quantity present in a sample of urine some precautions are necessary, for there are present with it in solution various other solids, both organic and inorganic, some of which would interfere with the process; for example, in Liebig's process the phosphates, sulphates, chlorides, and albumen must first be separated before commencing the analysis; and, even in the hypobromide of sodium method, creatin and traces of other extractives are decomposed with the urea, giving a result somewhat above the mark. Nevertheless in this secretion the urea exists in such large quantities (3 to 4 per cent.), being more than half the total solid residue, that results which are very accurate may still be obtained.

*Estimation in Blood.*—To estimate the amount of urea in the blood is far more difficult, and for this reason:—but a very small quantity is present (4 parts per 10,000), and this, a mere trace, co-exists with a mass of organic matter containing twenty times its weight of extractives alone. In these extractives many

azotised substances are found which are precipitated, and decomposed in a way similar to urea, which has therefore to be separated out first in a tolerably pure form before the quantity can be estimated. This separation from the blood is the difficult task to be overcome; the estimation of the quantity will then be an easy application of known methods.

Much has already been done towards obtaining a successful quantitative estimation of this substance in blood, and it may be well to review shortly one or two methods which have been tried.

Sir Robert Christison was one of the first pioneers in this direction, for he obtained urea from the blood of patients suffering from Bright's disease, in which it is greatly increased in amount. His method did not pretend to great accuracy; it was but a demonstration, and insufficient to show the crystals from normal blood, in which it was not then known to exist. He took the clear serum, concentrated it, and crystallised the urea out as a nitrate.

Joseph Picard's<sup>1</sup> method is one which has been more sharply criticised by more recent observers, and not without some reason; nevertheless, it has not been much improved upon. He mixed 100 cc. of blood at 96 per cent. with an equal volume of absolute alcohol, acidulated by a few drops of acetic acid, heated the mixture for a few minutes on a water bath, and pressed out the fluid through a calico filter; the process he then repeated with the residue, and evaporated the total filtrate rapidly on a water bath, and extracted the residue with alcohol, which he evaporated off. The residue is then mixed with ether one part and absolute alcohol two parts, filtered, evaporated, and the residue dissolved in water, from which much organic matter is precipitated by acetate of lead, the lead eliminated by a stream of sulphuretted hydrogen, the liquid concentrated, and the urea estimated by Liebig's process.

In criticising this method, I may state that (leaving out points of detail) during this long process a very large proportion of the urea must have decomposed, and from experience of this method I find it is impossible to get rid of many extractives which will

<sup>1</sup> De la présence de l'urée dans le sang, à l'état physiologique et à l'état pathologique.

all be estimated as urea, Liebig's process being the worst possible in this instance.

Dr. Richard Gscheidlen<sup>1</sup> has modified this method, adding, however, faults of his own. Blood is poured into boiling water, and sulphuric acid is added, drop by drop, until complete coagulation occurs. It is then filtered, the filtrate evaporated, and the residue extracted with alcohol, evaporated and dissolved in water. It is then precipitated with nitrate of mercury, and the mercury removed by a stream of sulphuretted hydrogen, evaporated and crystallised out as a nitrate; the crystals are dried and weighed. The method of drying and weighing is alone fatal to the process, for such a procedure is alone allowable when the substance is very pure, which is certainly not so in this case. I find, too, that the greater part of the substance is lost before the estimation is completed.

The only other method which it is desirable to notice is that of Professor Arthur Gamgee. He takes 50 grammes of blood and mixes it with twice its volume of absolute alcohol, and places it on one side for twenty-four hours. It is then filtered, and the filtrate evaporated, extracted with alcohol, dissolved in water, and estimated by Hüfner's method in a Dupré's apparatus.

One may accept the author's own criticism—which is, that it is an estimation of the nitrogen given off from all the alcoholic extracts of blood; but that, inasmuch as urea forms the chief bulk of these, one may roughly take it as indicating the quantity of urea itself.

Other methods have been proposed, but a more detailed account of these may be omitted; any improvement will probably be in the lines indicated by Picard and Gamgee. It will have been seen that the great difficulty lies in the fact that urea slowly decomposes into carbonate of ammonia, when a solvent containing it is evaporated; moreover, if organic impurities be present in the solution, this is greatly increased. I find that if defibrinated blood be evaporated on shallow dishes, taking every care that the temperature rises no higher than 50° cent., not a trace of urea is to be found in the hard cake which remains;

<sup>1</sup> *Studien über den Ursprung des Harnstoffs im Thier Körper.* Breslau, 1871.

nay, much pure urea may previously have been added with the like result.

In the alcoholic and watery solutions obtained by Picard and Gscheidlen fewer impurities are present; and although urea is found in the residue, it is diminished vastly in amount.

The first step taken in my investigations was to endeavour to diminish this loss, and every plan was tried which promised the least chance of success.

It was thought that possibly the high temperature used was prejudicial, and accordingly evaporations were conducted with solutions of a known strength, but at a temperature lower than one ordinarily uses. Flat porcelain dishes were used, and the temperature was kept below 50° Cent. Unfortunately this was of little avail, the loss remaining much the same as before. Failure also resulted when the urea solution (blood extract containing it) was evaporated at even a lower temperature, with diminished atmospheric pressure, nor was the acidification of the fluid with a few drops of acetic acid of any use. Urea, as has before been mentioned, forms well-defined salts with mineral acids; it was thought that by changing urea into a nitrate, or an oxalate, these might prove more stable, and as the ordinary methods admit of their estimation in these forms, the process might succeed, it being also always easy to reduce them as a last step into urea again. On evaporating an alcoholic solution of the oxalate (0.1 gramme per litre), there was found to be but 2 per cent. of loss, which is less than would follow if urea itself had been in solution. It seemed, therefore, that although an advance had been made by the use of the oxalate, yet some loss was inevitable, and that no method involving evaporation could be said to be perfectly accurate. It is still possible, however, to reduce this loss by using as little fluid as possible; and, above all, to obtain the urea unmixed with other organic solids, the presence of which is so prejudicial to the accuracy of the results. To separate the albumen from blood, either with alcohol or boiling acidulated water, means a dilution of the blood with several volumes of fluid, as we have already seen; worse than this, the alcoholic or watery extracts contain so many extractives that the urea passes through several processes, entailing evaporation before it is pure enough for final estimation. Can we

use less fluid and have fewer evaporations is the question before us?

Hearing that tungstic acid does not precipitate urea, and knowing also that it precipitates most organic substances, I tried to separate the urea by means of this reagent. Diluting with only two-third volumes, it was possible very completely to separate out the albumen as a fine granular mass by the addition of tungstate of soda and acetic acid. The after process was, however, very complicated, for the urea had to be separated from the tungstic acid, acetate of soda, and many extrac-tives, so that the loss was as great as ever.

*Separation by Dialysis.*—The separation by dialysis had long suggested itself to me as a possible method. It had not been tried, however, as it promised to be tedious. Hearing, however, from Professor Browne, that on placing blood in a dialyser, with alcohol in the outer vessel, it became quite dry in a few hours, an encouragement was given to a trial of this method. It must be borne in mind that urea is very soluble in alcohol, and would, therefore, pass into it with the water of the blood. A single trial proved this to be the case, and a few experiments sufficed to found the method, which will now be described.

*Method.*—80 cc. or 100 cc. of defibrinated blood are placed within a dialyser, so as to form a thin layer on the parchment paper (the dialysers that I use are made of glass, the inner vessel having a diameter of from eight to nine inches, the outer one-half or three-quarters of an inch more). In all cases the blood must not form a layer more than about 3 mm., otherwise the lower stratum will become dry and impervious, while the outer one will still remain fluid. 100 cubic centimetres of alcohol are then poured into the outer vessel, and the whole is covered. In from four to eight hours the alcohol in the outer vessel is seen to have risen, being increased in volume by the fluid parts of the blood, which blood forms now a dark tough cake within the dialyser, sticking, as a rule, to the parchment paper. Of course this still contains much urea, which must be washed out of it. For this purpose it is not sufficient to pour fresh water over it; it must be detached from the parchment paper, and bruised with the water in a mortar. The paper, with the blood adhering to it, is brought over a flat dish, gently

heated in a water bath, the blood being detached with the help of a little warm water. Fresh parchment is now placed over the ring of the dialyser, and the dialyser and the bruised mass brought into it and placed again in the alcohol, the blood soon becoming dry again from abstraction of water. The blood in the dialyser is now warmed to drive off the alcohol that is mixed with it, and the process of dialysing into fresh alcohol repeated. When water is added after the first time, the blood does not require to be taken from the parchment, for it can readily be mixed with the water by the help of a curved spatula. The two portions of alcohol are evaporated, and the residue extracted again with a little alcohol, the residue of which contains, as a rule, little else than urea. It is washed with petroleum naphtha,<sup>1</sup> which extracts fats and colouring matter, extracted with acetic ether, and the urea estimated. I accomplished this in Germany by a method introduced by Bunsen:—the urea is heated with an ammoniacal solution of chloride of barium in a sealed tube at a temperature of 200° Cent., decomposition of the urea takes place, carbonic acid gas and ammonia being formed, and the former is determined by weighing the carbonate of barium produced. Lately, I have used Hüfner's method, which, after a long trial and comparison with others, I can affirm to be without a rival. There is a loss with this process of above 7 per cent., for while by calculation one gramme of urea should yield 370 cc. of nitrogen at 0° Cent., and with 760 mm. of pressure, in practice only 340 cc. are obtained.

The loss is, however, wonderfully constant, and one may determine to one-third of a millegramme with great accuracy. On adding sugar to the urea, a suggestion for which I am indebted to Professor Gamgee, almost all the nitrogen is given off (363·4 cc.). As far as accuracy of result is concerned, however, the addition of the sugar is of no value, for one can always allow for the deficit. From the general remarks made at the beginning of this essay, it will be seen that the method is not a strictly accurate one. Evaporations were unavoidable, yet were reduced to a minimum, and the oxalate, a more stable compound,

<sup>1</sup> While investigating the solubilities of urea, seeking for some fluid in which it is insoluble, I found that this was the case with petroleum naphtha, by means of which it can be completely separated from fat and many extractives.

was dealt with. Above all, the urea was obtained from the first in a state of comparative purity, which, as we have already seen, is of great importance in the subsequent evaporation. Another advantage is, that no metallic salt is introduced, traces of these being prejudicial. The final estimation by Hüfner's method has special advantages over all others. In the first place, there is little chance of estimating other substances with the urea as in Liebig's process, and so increasing the results, for creatin is the only one which occurs in any quantity, and this is removed. In the second place, the process is accurate, and very easy of application. My method was, of course, tested with great care. Two portions of blood were taken, and to one was added a known quantity of pure, dry urea. Provided the method be perfectly accurate, the results of the two analyses should show a difference equal to the quantity of urea added. I found that there was a most distinct loss as might have been expected, but that it did not exceed 7 or 8 per cent., a far better result than obtained by other methods. Now, as part of this loss was constant, the error in a series of experiments would be represented by even a lower figure. By this method I have never failed to obtain a small naked eye demonstration of urea from so small a quantity of blood as 10 cc.

The experiments described above were conducted in the physiological laboratory of Professor Ludwig, who suggested to me this line of research. They occupied my time for over four months, the processes being for the most part very laborious and tedious.

It has long been a point much striven for, whether urea exists in muscle or not. Most entirely deny its existence there; a few chemists, on the other hand, maintain that they find it in large quantities (Picard the younger).

I have investigated the subject with my method of dialysing into alcohol, and find that it certainly does exist there, but in small amount. A dog or rabbit is bled to death, and the hinder legs are washed out with a very weak warm solution of common salt,  $\frac{1}{4}$  per cent. When perfectly bloodless, sufficient muscle is cut away, freed from fat and fascia, and a weighed portion, 80 to 100 grammes, taken. This is cut up into small pieces, and mashed in a mortar with glass and sand until a perfectly uni



form pulp is obtained. This is then stirred with a little water, and the fluid parts squeezed through a linen cloth, and the dry mass left behind treated two or three times in the same way. The watery extract is now placed upon the parchment paper of a dialyser, and the watery parts rapidly pass into the alcohol. The latter is now evaporated, and a brown mass is left, which contains, besides the urea, some creatin in a small quantity. The muscle residue is washed once or twice with fresh water until all the urea has passed out into the alcohol. The residue is then washed with petroleum ether, re-extracted with alcohol, in which creatin is practically insoluble. It is then extracted with acetic ether, in which creatin is insoluble. A straw-coloured mass is left on evaporating the ether, out of which the urea crystallises in two or three days if it be placed over sulphuric acid. In some preparations this is very slow, and but faint indications of the crystals are to be seen. It is estimated by the hypobromide of sodium method. It is undoubtedly urea with which we have to deal, for I know of no crystalline organic compound which would pass through these various extractives. The crystals, too, are characteristic; still more so those of nitrate of urea formed by the addition of a drop of pure nitric acid. The average quantity I find to be 0.01 per cent., which is, practically, but a trace. According to the statements of Picard and others, a far greater amount than this is to be found. That this is false I have fully proved by adding beforehand to my muscle extract a few milligrammes of pure urea. The addition was at once detected in the result, showing that no great loss could have occurred. An examination of Picard's method shows that he estimated not only the urea, but a large quantity of extractive (principally creatin) as well. Urea is mixed in muscle with a number of other extractives, from which it is difficult to separate it; hence the statement that it does not exist there, or, if it be not properly separated, they are estimated with it, and apparently swell the amount to an absurd degree.

I have sought with these methods at my disposal to investigate the variations in the amount of this substance present in blood and in muscle. The former subject has received considerable attention, but very little reliable information has yet been obtained, one observer flatly contradicting his prede-

cessor, almost the only fact known being that in Bright's disease it is greatly increased in amount. As regards the variations of this substance in muscle of course absolutely nothing is known, there being up till now no method for its extraction. The results of the younger Picard (in the *Compt. Rend.* for 1878) may safely be set on one side.

The first problem, the solution of which I have attempted, was whether or not urea is increased in blood and muscle during muscular activity. In this paper no reference will be made to experiments which did not succeed, owing to failure of analysis or to some other cause; there were naturally many such.

*Experiment.*—A large Newfoundland dog was deprived of food for twenty-four hours. It was then bled to death; the blood was defibrinated, and a portion reserved for analysis (A). The rest was injected into the arteries of the hinder limbs, collected as it flowed back through the veins, and passed again and again through the vessels, in all no less than thirteen times. During this process the muscles were tetanised powerfully by an interrupted current, the experiment lasting over forty minutes. A portion of this blood, which we may call B., was then taken, and the urea estimated.

(A.) Blood first drawn contained 0·021 per cent. of urea.

(B.) Blood passed through tetanised by 0·022 per cent. of urea.

There is, as will be seen on comparing the two, practically no difference between them. The method is open, however, to some objections, for there may be a very material production of urea in the muscle without our being able to detect it after passing the blood so few times through the limb. Were the method one of great exactitude, which, alas!—in common with all other methods for the estimation of the various constituents of blood—it is not, then no doubt the question could with certainty be settled. Unfortunately, however, in chemical physiology we as yet possess few methods which give, chemically speaking, anything like rigorously accurate results. I viewed the experiment then as one which, while it might corroborate another, yet could not give a decisive answer to our question.

*Experiment.*—A young sheep dog, which for forty hours had been deprived of solid food, was exercised in a large tank of water until muscular exhaustion was well marked. It was killed thirty minutes after, and the urea estimated in 100 c.c. of the blood. A very small quantity was found—only 0·0186 per cent.

*Experiment.*—In this case the subject of the experiment was a young terrier dog, which had fasted for seventeen hours. The muscular exhaustion was quite as marked as in the former case, with almost as small a quantity of urea in the blood. 100 c.c. of blood contained 0.020 grammes of urea. 100 grammes of muscle contained 0.0085 of urea.

These three experiments, I believe, show conclusively that urea is not formed to any extent in muscle, or in any other part of the body during muscular exercise. The last experiment in which an estimation of the urea in muscle was made, is, of course, not conclusive; but the fact that it is not increased at all—being rather below the normal—in the blood is a most strong corroboration. Urea being so diffusible, it is almost certain that it would pass into the blood from the muscle were it there in any great amount. It may be added that, as far as the experiments and subsequent analyses were concerned, nothing more successful could have been wished, no hitch whatsoever occurring. A very curious fact, and one which at this period of the investigation much perplexed me, is the following:—When in Germany I made several urea estimations, giving always a high percentage (above 0.03 per cent.), but in England I obtained always lower results, such as are typified by the afore-described experiments. It at last struck me as the animals had been differently fed, the discrepancy might be due to this. In Germany the dogs were always killed two or three hours after a meal, while in England they received no food for at least fifteen hours previously. It was obviously of importance to find out whether the time after food had any influence upon the amount of urea in the blood.

*Experiment.*—Two rabbits were selected as nearly as possible alike in all respects. One was killed five hours after a full meal of carrots; the urea in the blood estimated, with the result that 0.0486 per cent. was found. The other was kept for thirty-six hours without food, and 0.0372 per cent. of urea was found on analysis. There is obviously one objection to this, and it is that one cannot be sure that you are dealing with two animals exactly alike in all respects save the time they were killed after a meal. As it is impossible to obtain from a rabbit enough blood to make two estimations, I then selected a number of large dogs, from which the abstraction of 100 c.c. of blood may be made without interfering much with the condition of the animal.

*Experiment.*—A full-grown fat sheep dog was kept without food for

forty-eight hours, at the end of which time 50 c.c. of blood was drawn from a vein, and on estimation was found to contain 0.038 per cent. of urea. Two pounds of boiled steak were then given it, and four hours after it was bled to death. The blood contained as much as 0.064 per cent., showing an increase of 68 per cent. on the first portion.

*Experiment.*—This was a repetition of the former experiment upon a fair-sized young Newfoundland dog. 50 c.c. of blood taken from the animal after abstinence for forty-eight hours contained 0.027 per cent. of urea. It was then fed with two pounds of boiled steak, after which the percentage increased, being 0.041 when killed four hours afterwards. There was again an increase of about 52 per cent.

These three experiments show that if to an animal which has for some time fasted, proteid food be given, the urea is increased by a very great amount in the blood. As it is just possible, however, that the abstraction of the 50 c.c. of blood which were drawn from the animal in the fasting condition, might of itself produce some change or other in the system, it was thought advisable to reverse the experiment.

*Experiment.*—A large dog was fed with two pounds of boiled steak, and four hours after 50 c.c. of blood were withdrawn and found to contain 0.036 per cent. of urea. It was then kept without food for forty-eight hours, killed, and the blood analysed. The urea was now found to exist only in a very small quantity—0.019.

This last experiment, in conjunction with those previously detailed, shows conclusively that urea is found in large amount during and after the digestion of proteid matter, and that if no further increase to food be given, the quantity gradually falls, the greater part being probably eliminated by the kidneys. Where the formation of urea occurs is a question we shall not touch from a literary point of view; for, were the slightest digression made, fields of controversy would be opened up so vast, that their exploration would be quite out of the scope of the present essay. A curious experiment was then undertaken to solve the following question—Can urea be formed from peptonised albumen when injected into the blood?

The importance of this is that albumen is absorbed into the system as peptone, where it is mostly at any rate converted again into albumen. If now we inject peptone into the system, it will also be reconverted into albumen with the formation of

urea, just as in the absorption of proteid matter from the stomach and intestines, provided the formation of urea occurs in the liver or other glands of the body, and not during its absorption through the cells of the villi, as some have thought.

*Experiment.*—A dog which had been fed previously was bled to the extent of 50 c.c. The blood contained 0.064 per cent. of urea. Some peptone solution (Darby's fluid meat) was injected into the femoral vein. Most curious symptoms were then observed; for, when but a drachm or two of the solution had been given, the dog became most uneasy, struggling violently. I injected a little more (two drachms), when the animal went into violent convulsions.<sup>1</sup> The heart was found, on a *post-mortem* examination, to be flaccid and full of blood; and the lungs, liver, and kidneys—indeed, all the internal organs—were engorged. 50 c.c. of the blood contained 0.068 per cent. of urea.

This small increase cannot be relied upon as showing any certain results as far as the formation of urea from the peptone is concerned. The dog was thrown into so abnormal a condition that the experiment must be discarded. I may mention that I have tried since the injection of peptone into a dog to know if these curious results are constant. It was not carried on beyond the first stage of great uneasiness, but the symptoms were the same as far as the experiment went.

*Experiment.*—Two similar large white rabbits were kept for some days in the same cage, having exactly the same food. Rabbit (A.) was then deprived of food for twenty-four hours, when the blood was found to contain 0.082 per cent. of urea. Rabbit (B.) was kept without food for thirty-six hours—half again as long as the other—and four drachms of strong peptone solution was injected into the femoral vein, and the animal was killed half an hour after; 0.0936 per cent. of urea was found.

This indicates an increase of the urea after the injection of the peptone. A curious point is that you can inject a large quantity of peptone solution into the veins of a rabbit without producing any external symptoms whatever, except that the animal stops breathing for a little time during the injection. It does not seem to be distressed at all.

I may sum up shortly the result of these experiments.

(1.) A large quantity of urea is found in the blood of an

<sup>1</sup> The dog was under the influence of chloroform, and insensible.

animal during and shortly after digestion ; far less if it be in a starving condition.

(2.) The injection of a solution of peptone also produces an increase in the amount of urea, although this point requires further investigation.

(3.) Urea is not increased in blood during and after muscular exercise to any appreciable extent.

(4.) The amount may vary greatly in various conditions (as above) of the healthy state ; in my own cases this may be from 0.095 to 0.010 per cent. It is impossible to give an average unless the time after food be taken into consideration.

(5.) Urea exists in muscle in small amount (about 0.010 per cent.).

(6.) There is no increase perceptible as the result of muscular activity. Probably the quantity existing in muscle varies with, and depends upon, that in the blood, a small and varying quantity naturally diffusing into it.

I have avoided purposely stating any views of my own as to where urea is formed in the body, and I have kept clear of any controversy, and for this reason especially, that I feel no generalisations can be made before much more extended observations have been carried out. My own work on this subject has, in the meanwhile, been suddenly brought to an end by my failure to obtain a licence to perform so simple an experiment as the bleeding of a dog under chloroform ; but having been urged to publish any results, I do so with the full consciousness of their want of completeness.

It is with pleasure that I acknowledge assistance in the form of a grant of £25 which I have received from the Scientific Grants' Committee of the British Medical Association, my chief expenditure being on dogs and absolute alcohol. My friend and former teacher, Professor Rutherford, has given me every facility which his laboratory can afford, and it is with gratitude that I acknowledge his kindness. I am fortunate also in the possession of a friend in Professor Gamgee, of whose great knowledge of this branch of chemical physiology I often availed myself.

ON THE HOMOLOGIES OF THE LONG FLEXOR  
MUSCLES OF THE FEET OF MAMMALIA, WITH  
REMARKS ON THE VALUE OF THEIR LEADING  
MODIFICATIONS IN CLASSIFICATION. By G. E.  
DOBSON, M.A., M.B. (PLATES IV., V., VI.)

WHILE studying the anatomy of the Insectivora I was much struck with the many modifications of form and arrangement presented by the long flexors of the feet in different species, and careful examinations of the nature of these modifications, and comparisons with those observable in the species of other Mammalian orders (which I have fortunately been enabled to carry out with considerable detail<sup>1</sup>) have demonstrated forcibly that the homologies of these muscles are very imperfectly understood by anatomists generally.<sup>2</sup>

In most species of mammals three long flexor muscles form the deep layer on the posterior aspect of the leg; these are—

1. *Flexor digitorum fibularis* = *Flexor hallucis longus* (Human Anatomy).

2. *Flexor digitorum tibialis* = *Flexor digitorum longus* (Human Anatomy).

3. *Tibialis posticus*.

These three muscles are represented by their homologues in most mammals; the *flexor fibularis* exists in all, and exhibits but slight modifications, but the other two are subject to much variability, and may be severally or collectively absent; the *flexor tibialis* especially varies very considerably within certain limits, so much so, indeed, that not being recognised under its altered conditions, its apparent or real disappearance has been

<sup>1</sup> Owing chiefly to the kindness of Prof. W. H. Flower, F.R.S., and Mr. W. A. Forbes, Prosector to the Zoological Society, who afforded me every opportunity for the examination of the valuable series of specimens in their charge.

<sup>2</sup> At the commencement of his paper "On the Disposition of the Deep Plantar Tendons in Different Birds," the late Professor A. H. Garrod remarks:—"The arrangement of the tendons in the palm of the hand and the sole of the foot among the Mammalia is a subject of great intricacy, as may be inferred from the comparison of the dissections of different animals."—*Proc. Zool. Soc.*, 1875, p. 339.

attempted to be explained by anatomists by supposing it to have undergone fusion with the *flexor fibularis*, thus forming a *flexor digitorum communis*,<sup>1</sup> while the differentiated muscle (unknown as such) has been described as part of the *tibialis posticus*<sup>2</sup> or referred to under the names of *tibialis posticus accessorius*,<sup>3</sup> *secundus*, or *internus*.<sup>4</sup> It is the chief object of this paper to demonstrate the true relations of this muscle, and to show that neither of the above supposed conditions really obtain in any of the species in which they have been described, nor probably in any species of mammal.

Of all the orders of Mammalia the Insectivora present by far the greatest amount of variety in the arrangement of the long flexors of the feet, and there is scarcely any modification of these muscles in the legs or feet of the species of other orders, which is not either represented by or capable of being derived from corresponding structures in the species of this small group, another indication added to the many of the central position of this order among those of the higher mammals.

<sup>1</sup> "The *flexor longus digitorum pedis* and the *flexor longus pollicis* are united," Owen, "Anat. of *Dasyprocta sexcinctus*," *Proc. Zool. Soc.*, 1832, p. 138. "*Flexor hallucis longus* pariter cum *flexore digitorum longo*," Hyrtl, *Chlamydomorphi truncati Anat.*, *Denkschr. Akad. Wissensch.*, Wien., 1855, p. 41. "*Flexor digitorum longus*, in the Wombat, is in reality a compound of two parts, the *flexor digitorum* and the *flexor hallucis*. In *Macropus giganteus*, *M. bennettii*, *Sarcophilus*, the Opossum, and Phalanger, these muscles are similarly fused," Macalister, *Ann. Mag. Nat. Hist.*, v. (1870). "The *flexor hallucis* and *flexor perforans* have a common tendon" (in *Erinaceus*), Huxley, *Anat. Vertebr. Anim.*, p. 446 (1871). "*Flexor hallucis longus* and *flexor communis digitorum* are conjoined," Murie, *Trans. Linnean Soc.*, xxx. p. 101 (1874). "The *flexor longus digitorum* and the *flexor hallucis* are inseparable" (in *Phascogale cinereus*), Young, *Jour. Anat. Phys.*, xvi. p. 237 (1882), and in the writings of these and other anatomists elsewhere.

<sup>2</sup> "Fasciculus separated from the *tibialis posticus*," Owen, *l.c.*; Cuvier, *Myologie*, pl. 268; Schulze, *Zeitschr. für Wissensch. Zoologie*, Leipzig, 1867, p. 13.

<sup>3</sup> "Infra Poplitei insertionem, novus exurgit musculus, qui Tibialem posticum viæ comitem laborisque socium legit. Retro malleolum internum in teretem fatiscit tendinem, qui margini pedis externo appropinquat, et ad basin ossiculi illius depressi finem asequitur, quod metatarso hallucis interne adjacet. *Tibiale posticum accessorium* non invite nominarem."—Hyrtl, "Chlamydomorphi truncati Myologia," *l.c.*

<sup>4</sup> Galton, *Trans. Linnean Soc.*, xxx. p. 558 (1869); Huxley, *l.c.*; Murie, *l.c.*; Macalister, *Introd. Syst. Zool. Morph. Vert. Anim.*, p. 304 (1878); Dobson, *Monograph of the Insectivora, Systematic and Anatomical*, pp. 34 and 57 (1882); Young, *l.c.*



We may, therefore, take the leading modifications of these muscles in Insectivora as a convenient basis for comparison, and, having described and figured them, pass on to the consideration of the principal varieties of form and arrangement presented by corresponding parts in the species of other orders.<sup>1</sup>

In *Centetes ecaudatus*, the common ground-hog of Madagascar, and the largest of living Insectivora, we have what may be termed the typical Mammalian leg and foot (Plate IV., fig. 1), for out of it might, by modification, be constructed the corresponding parts of the hinder extremity of any other known mammal. In the leg we find a distinct tibia and fibula, a tarsus of seven bones, five metatarsals, a hallux with two, and four digits with three, phalanges each. Of the muscles attached to these bones there are, besides extensors and the superficial flexors of the foot entering into the formation of the tendo achillis, the following long and short flexors, and other muscles of the plantar surfaces:—

1. *Flexor digitorum fibularis* (f.f.) = *Flexor hallucis longus* (Human Anatomy).
2. *Flexor digitorum tibialis* (f.t.) = *Flexor digitorum longus* (Human Anatomy).
3. *Tibialis posticus* (t.p.).
4. *Flexor digitorum brevis* (f.b.).
5. *Flexor accessorius* (f.a.).
6. *Flexores digitorum breves vel interossei*.
7. *Adductores hallucis, indicis, minimi digiti*  $a^1 a^2 a^3$ .
8. *Abductor ossis metatarsi minimi digiti* (ab. o. m.).
9. *Lumbricales*.

With the single exception of the first named, these muscles may, as already remarked, be severally or collectively absent until we find the limit reached in the marsupial genus *Hypsiprymnus* (and probably in *Chæropus* also), where, omitting the muscles forming the tendo achillis, the long flexors are represented by the *flexor fibularis* only, and the plantar muscles are altogether wanting (see Plate V., fig. 9).

<sup>1</sup> The determination of the homologies by means of the nerve supply, so excellent a guide in many cases, cannot be successfully applied here, as all three muscles under consideration are supplied by the same nerve, namely, the posterior tibial.

In *Centetes ecaudatus*, however, they are severally and collectively well developed, and the long flexors have the following origins and connections:—

1. *Flexor fibularis* arises from the head and greater part of the posterior surface of the fibula, and, forming a moderately large tendon, passes into the foot through the deep groove on the inner side of the os calcis, becoming flattened out and connected with the superficial surface of the similarly expanded tendon of the *flexor tibialis*, before dividing into slips for the three middle toes (Plate IV. fig. 1, *f. f.*).

2. *Flexor tibialis* arises in close connection with the fibular flexor from the head of the fibula and part of the shaft of that bone, also from the interosseous membrane and part of the adjacent tibial margin, and, forming a more slender tendon, passes through a groove on the posterior surface of the internal malleolus, and, spreading out, becomes superficial to and united by its deep surface with the expanded tendon of the fibular flexor, but sends off, laterally, distinct slips for the hallux and fifth toe (Plate IV. fig. 1, *f. t.*).

Although the fibular and tibial flexors are, from their relative positions in the leg and foot, undoubtedly homologous with the *flexor hallucis longus* and *flexor digitorum longus*, respectively, of Human Anatomy, yet, as we have above-noted, their ultimate distribution in the foot is very different, that muscle which is homologous with the flexor of the hallux in man being here distributed to the three middle toes, whilst the homologue of the flexor digitorum supplies the hallux and fifth toe.

In the species of the allied genera *Hemicentetes* and *Ericulus* the relations of the tendons of these muscles in the foot are quite similar, and in those of *Oryzorictes* and *Microgale* (with united and rudimentary fibula), which belong to the same family, although the superimposed tendons are more closely united, the tendon for the hallux is still distinctly seen to be given off by the flexor digitorum.

3. *Tibialis posticus*, a much smaller muscle; arises in connection with the tibial flexor from the head of the fibula and interosseous membrane, but chiefly from the fibular side of the tibia under cover of the popliteus, and for a short distance below the lowest point of insertion of the latter muscle, forms a strong tendon

which passes down the postero-internal margin of the tibia, and, crossing the malleolus in a separate groove on the tibial side of the groove for the tendon of the *flexor tibialis*, is inserted into the scaphoid bone (Plate IV. fig. 1, f. f.).

Such are the origins and attachments of these three muscles in *Centetes ecaudatus*, with which we proceed to compare those of the corresponding parts in other mammals. Before describing the modifications in origin it will be necessary, however, to refer to the insertions in order to gain a clear idea of their homologies.

In Plate IV., figs. 2 and 3, representing the long flexor muscles on the posterior aspect of the leg and plantar surface of the foot of *Solenodon cubanus* and *Erinaceus europæus*, respectively, two distinct arrangements of the tendon of the *flexor tibialis* (*flexor digitorum longus*) are shown:—

- a. Tendon of the *flexor tibialis* united with that of the *flexor fibularis* (*flexor hallucis longus*) (fig. 2).
- b. Tendon of the *flexor tibialis* completely separated from that of the *flexor fibularis* (fig. 3).

These two conditions represent, as will be shown hereafter, the leading modifications of these muscles throughout the Mammalian series.

In Plate IV. fig. 2, f. t., the tendon of the *flexor tibialis* (*flexor digitorum longus*) is seen passing through a groove on the tibial side of the internal malleolus accompanied by that of the *tibialis posticus*, which goes to its insertion into the scaphoid bone, while the former divides into two tendons, one of which is connected with the *flexor fibularis*, the other is inserted into the tibial side of the first metatarsal bone.

That the muscle marked *flexor tibialis* in fig. 2 is homologous with those similarly marked in figs. 1 and 3, admits of no doubt. In figs. 2 and 3 its origin is seen to be wholly removed from the fibula, but intermediate conditions are common in many species, and very well illustrated in the Marsupials in *Cuscus maculatus* and *Belideus flaviventer* (Plate V. fig. 8, f. t.). In *Erinaceus europæus* (Plate IV. fig. 3, f. t.), although the fibular slip from its tendon is no longer connected with that of the *flexor fibularis*, the tibial slip maintains the attachment to the first metatarsal bone as in *Solenodon cubanus* (fig. 2). In the Edentate *Orycteropus capensis* the *flexor tibialis* arises as in *Centetes ecaudatus*,

but divides after crossing the malleolus into two tendons, as in *Solenodon cubanus*, one going to the side of the *flexor fibularis*, the other to the sesamoid ossicle lying alongside the ento-cuneiform bone.<sup>1</sup> This condition is, therefore, intermediate between that of *C. ecaudatus* and *S. cubanus*. On the other hand, the fibular slip may alone remain connected with the superficial fascia and integument of the sole of the foot, as in *Gymnura rafflesii* and many species of *Erinaceus*.

In all the above cases the *tibialis posticus* is well developed; its tendon passes down generally under cover of that of the *flexor tibialis* when crossing the malleolus, and is inserted into the scaphoid or ento-cuneiform bones.

Having thus determined that this muscle, hitherto considered by anatomists as forming part of a double *tibialis posticus*, and named variously *tibialis posticus accessorius*, *secundus*, or *internus*, must really be looked upon as the representative of the tibial flexor of the toes (*flexor digitorum longus*) of *Centetes ecaudatus*, *Solenodon cubanus*, and many other species, we are in a position to consider its various leading modifications in origin and attachment in the species of Insectivora and other mammals.

*Flexor tibialis* may, by increase in the size of the *flexor fibularis*, have origin wholly from the tibia, as in *Gymnura rafflesii*, *Erinaceus europæus* (Plate IV. fig. 3), &c., or, as in *Condylura cristata* and *Myogale pyrenaica*, the fibular flexor may have such an extensive origin from both bones that the origins of the *flexor tibialis*, as well as of the *tibialis posticus*, may both be superficial from the heads of the tibia and fibula, and their tendons pass down on the surface of the much larger muscle (Plate IV. fig. 5, *f. t.*, *t. p.*); or, more rarely, the muscle may be altogether absent. On the other hand, owing to increased functions, as a supplemental flexor of a large and specialised hallux, as in *Cuscus* and *Belideus*, where the fibular flexor is also largely developed, it may take origin from both bones, but principally from the tibia.

Its variable condition when divorced from connection with the fibular flexor, and from its office as joint flexor of the toes, is strikingly illustrated, even within the limits of a single genus,

<sup>1</sup> The connections of this muscle are so given by Galton (*l.c.*, p. 597), who, however, calls it the *tibialis posticus*, which really appears to be altogether absent in this as in many other species of mammals to be referred to farther on.

as in *Erinaceus*. In *E. Europæus* this muscle sends off both a fibular and a tibial slip (Plate IV. fig. 3). This is probably due to the comparatively large size of the hallux in this species. In all the other species possessing this muscle (of which the anatomy has been examined by the writer), namely, in *E. grayi*, *macracanthus*, *niger*, and *jerdoni*, its tendon goes only to the superficial fascia and integument of the sole of the foot: in *E. albiventris*, *deserti*, *diadematus*, and *pictus* (and probably in *E. micropus* also), in all of which the hallux is very small or wanting, and the plantar callosity small or obsolete, this muscle is absent (Plate V. fig. 6). In other species of Insectivora the arrangement of the tibial flexor is as follows:—All the species of *Soricidæ* and *Talpidæ* examined had this muscle separated in the foot from the fibular flexor, as in the *Erinaccidæ*; in *Sorex* (Plate IV. fig. 4), it arises from the head and internal margin of the upper extremity of the tibia, and the tendon, passing down in the same groove with and superficial to that of the *tibialis posticus*, is inserted into the sesamoid ossicle lying on the tibial side of the ento-cuneiform bone, and through it is connected by a tendon with the tibial side of the base of the first phalanx of the hallux; in *Talpa*, *Condylura*, and *Myogale* the arrangement is similar. On the other hand, *Galeopithecus*, *Tupaia*, *Macroscelides*, *Potamogale*, *Chrysochloris* and *Solenodon* conform to the *Centetes* type. In *Galeopithecus philippinensis* the *flexor tibialis* arises from the tibia as high up as the insertion of the *popliteus*, and forms (in the leg) two tendons, the superficial becomes external, and sends a slip to the other tendon in the foot; both tendons, however, unite before coalescing with the superficial surface of the tendon of the fibular flexor, but the tendon for the hallux is first given off. In *Tupaia ellioti*, as in the species of *Chrysochloris*, a flat sesamoid bone is formed in the tendon of the flexor fibularis, where it glides across the os calcis; the *flexor tibialis* arises from the greater part of the posterior surface of the tibia from its head downwards, forming a tendon nearly as large as that of the flexor *fibularis*, with which it unites superficially. In *Potamogale velox* the tibial flexor arises from the interosseous membrane and adjoining margin of the tibia, under cover of the large popliteus, and develops a very slender tendon, which, passing down in the same groove with the *tibialis posticus*, is inserted into the

side of the large fibular flexor. The origins and connections of this muscle in the different species of the family *Centetidae* closely agree with those described above in *Centetes ecaudatus*. In *Chrysochloris* the *flexor fibularis* is large, arising from the greater part of the fibula, and forms a strong tendon, which, receiving on its deep surface the tendon of the *flexor tibialis* immediately before crossing the ankle joint, develops a sesamoid bone, where it lies in the groove on the os calcis, and divides into tendons for the five toes. *Flexor tibialis* is well developed, arising from the back and inner side of the upper third of the tibia, internal to the insertion of the short *popliteus*, and forms a strong tendon, which, owing to the position of the origin of the muscle from the very anteriorly placed tibia, in passing down lies close to the united tibia and fibula, and, consequently, in going to join the fibular flexor at the ankle joint, curves outwards in front of the tendon of the more posteriorly placed *tibialis posticus*, and unites with the deep surface of the tendon of the former muscle.

*Tibialis posticus* is much less variable; in all the species of Insectivora, except in those of *Myogale*, where its tendon unites with that of the *flexor tibialis* above the malleolus (Plate IV. fig. 5), it may be described as arising from the heads of the fibula and tibia and adjacent interosseous membrane, being more or less concealed between the fibular or tibial flexor and the *popliteus*, forming a rather slender tendon, which passes down either in the same groove on the internal malleolus with, and under cover of, the tendon of the *flexor tibialis*, or on its tibial side, and is inserted into the scaphoid, or ento-cuneiform, or, rarely, as in *Chrysochloris*, into the base of the first metatarsal bone. Some of its fibres are often more or less connected with those of *popliteus*.

With these preliminary remarks on the modifications of the long flexor muscles of the feet in Insectivora, we proceed to consider their structure and arrangement in other Mammalian orders.

#### MONOTREMATA.

*Flexor fibularis* arises, in *Echidna setosa*, from the head and greater part of the posterior surface of the fibula, forms a strong

tendon, which receives on its outer edge in the foot the fibres of a large *flexor accessorius*, and divides into three tendons only for the three inner toes, the two outer toes being supplied by the *flexor brevis* only (Plate V. fig. 7, *f.f.*).

*Flexor tibialis* arises from the inner side of the head of the fibula, and forms a moderately large tendon, which crosses the back of the internal malleolus over the tendon of the *tibialis posticus*, and is inserted into the ungual phalanx of the hallux by a broad expansion (fig. 7, *f.t.*).

In *Ornithorynchus paradoxus* this muscle, according to Meckel,<sup>1</sup> contains in its tendon a sesamoid bone, and is inserted into the first phalanx of the hallux.

*Tibialis posticus*, more than double the size of the *flexor tibialis*, arises from the head of the fibula under cover of that muscle and from half the shaft of the bone, and, forming a thick tendon, passes across the malleolus under cover of the *flexor tibialis* tendon to its insertion into the scaphoid bone (fig. 7, *t.p.*).

#### MARSUPIALIA.

*Belideus flaviventer*.—*Flexor fibularis* (Plate V. fig. 8, *f.f.*) arises from almost the whole length of the fibula; about the middle of its posterior surface a superficial part (*f.p.*) separates from the mass, and divides on crossing into the foot into three tendons, which form the perforated tendons for the three outer toes; of these the middle is the largest, and the outer and inner, especially the latter for the third toe, very slender; the perforated tendon for the second toe is derived from the calcaneum; the main tendon divides into five for the five digits, the second and third being very slender.

*Flexor tibialis* (fig. 8, *f.t.*) arises by a fibular and tibial head, the former smaller than the latter; these unite in the upper third of the leg, and pass down in a long tendon, which, crossing the malleolus internally, is inserted into the ento-cuneiform bone and into the sesamoid bone lying on its fibular side.

*Tibialis posticus* (fig. 8, *t.p.*) is exceedingly small, arising under cover of the fibular head of the preceding from the head of the fibula; its very slender tendon passes down under cover of that

<sup>1</sup> *Anat. Comp.*, French ed., vi. p. 425.

of the preceding, and is inserted into the capsular ligament of the calcaneo-scapoid articulation and into the scaphoid bone.

*Acrobates pygmaeus*.—In the Opossum Mouse the *flexor fibularis* is large, arising from the greater part of the fibula, interosseous membrane, and adjoining margin of the tibia, and, passing into the foot, divides into slips for the five toes. *Flexor tibialis* is much larger than the *tibialis posticus*; it arises from the head and upper fourth of the tibia under cover of the edge of the *popliteus*, and, forming a moderately strong tendon, passes down along with that of the *tibialis posticus*, and is inserted into the internal cuneiform bone. *Tibialis posticus* very small, with an exceedingly slender tendon, arises from the upper part of the interosseous membrane, and from the adjacent sides of the heads of the tibia and fibula, and is inserted into the scaphoid bone.

*Phalangista vulpina*.—*Flexor fibularis* arises from almost the whole posterior surface of the fibula, and forms a thick tendon, which divides into slips for the five toes. *Flexor tibialis* is much smaller, arising from the heads of the fibula and tibia, from part of the interosseous membrane, and from the fibular side of the tibia; its tendon passes down in a groove on the inner malleolus, and is inserted into the ento-cuneiform bone and into the sesamoid bone in the tendon of the plantaris, which plays over the scaphoid tubercle. *Tibialis posticus* is small, arising in intimate union with the *fibular flexor* from the head of the fibula, and from the tibial margin of that bone, also from part of the interosseous membrane, and is inserted into the scaphoid bone, its slender tendon passing down under cover of the *flexor tibialis*.

*Phascolarctos cinereus*.—In the Koala the *flexor fibularis* is very large, arising from almost the whole length of the fibula and part of the interosseous membrane, and forming a strong flat tendon, which gives off in the foot five slips for the five toes. Some of the superficial fibres in the leg separate from the general mass as in *Belideus flaviventer*, and divide it into two heads, from each of which a pair of slender tendons pass down into the foot, and form the perforated tendons for the four outer toes. *Flexor tibialis* is much smaller, arising from the head of the fibula, and from part of the interosseous membrane and surface of the *pronator tibiae* (*popliteus*) muscle; its tendon passes down in a



groove on the tibial side of the internal malleolus, and divides after entering the foot into two flattened tendons connected by an aponeurosis, of which the inner is inserted into the entocuneiform bone, the outer into the side of the sesamoid bone at the base of the first metatarsal. *Tibialis posticus* is very small, arising deeply between the preceding muscles from the head of the fibula and adjoining margin of the interosseous membrane, its very slender thread-like tendon passes into the foot in the centre of the space between the tendons of the fibular and tibial flexors, and is inserted into the calcaneo-scaphoid ligament.

*Cuscus maculatus*.—The arrangement of the long flexors is very similar to that of *Belideus flaviventer*, the only difference of importance being that the fibular head of the *flexor tibialis* is smaller, and the tendon of the muscle is inserted into the base of the metatarsal bone of the hallux.

*Phascolomys fossor*.—In the Wombat the arrangement of the long flexors are also very similar to that of *B. flaviventer*, the chief differences consisting in the continuation of the superficial slip of the *flexor fibularis* as a muscular body into the foot, where, as the *flexor digitorum brevis*, it gives off the perforated tendons for the three middle toes, none going to the fifth digit. *Flexor tibialis* arises mainly from the heads of the fibula and tibia, being scarcely connected with the shaft of the latter bone, which is occupied by the insertion of the *pronator tibie* (*popliteus*) muscle, and is inserted into the metatarsal of the rudimentary hallux. *Tibialis posticus* is quite similar to that of *Cuscus maculatus*.

*Perameles nasuta*.—*Flexor fibularis* is of great size, arising from the greater part of the fibula, interosseous membrane, and tibia below the insertion of the *popliteus*. The fibular and tibial parts are divided by a groove, in which lies the upper half of the extremely slender tendon of the *tibialis posticus*. These parts form strong tendons, which unite into one large tendon above the ankle joint, which, passing into the foot, divides into three parts—two large for the fourth and fifth toes, and a slender one (which again divides into two) for the rudimentary second and third toes; no tendon goes to the rudimentary hallux. *Flexor tibialis* is altogether absent. *Tibialis posticus* is reduced to the smallest possible size; its muscular part (not larger than

a barley-corn) arises, under cover of the *popliteus*, from the tibial side of the head of the fibula, and forms a thread-like tendon, which lies concealed for half its length in the deep furrow between the tibial and fibular heads of the *flexor fibularis*, then emerging abruptly, and running down on the surface of the tendon of the fibular head, it crosses the internal malleolus in a shallow groove, and is inserted into the scaphoid bone.

Many anatomists would, no doubt, consider the tibial part of this muscle (which, as above described, sends down a tendon to join that of the fibular part above the ankle joint), as the *flexor tibialis*, fused in its muscular part with the *flexor fibularis*; but the fact that its tendon, instead of crossing the *tibialis posticus*, is crossed by the tendon of that muscle, and, moreover, instead of entering the foot in a groove on the back of the internal malleolus, as in all other species, enters it in union with the tendon of the *flexor fibularis*, proves that it is really but the tibial head of that muscle, and that the *flexor tibialis* is altogether absent. This will be additionally demonstrated further on to be actually the case.

*Antechinus swainsoni*.—*Flexor fibularis* is large, arising from the greater part of the fibula and part of the interosseous membrane, and forms a thick tendon, which, passing into the foot, divides into four slips for the four outer toes. *Flexor tibialis* is small, arising from the head of the tibia on its fibular side and from part of the shaft of the bone, forms a long tendon, which glides over the back of the internal malleolus, superficial to that of the *tibialis posticus*, and spreads out into an aponeurosis, from which is given off the flexor tendon of the hallux and the perforated tendons for the other toes, being connected by a narrow fascial band with the tibial side of the *flexor fibularis*. *Tibialis posticus* arises under cover of the preceding from the head of the fibula in connection with the *flexor fibularis*, which, further down, separates it from the interosseous membrane, and, forming a long tendon, crosses the back of the internal malleolus under cover of the *tibialis posticus*, and is inserted into the scaphoid bone.

*Dasyurus macrurus*.—*Flexor fibularis* arises from the greater part of the fibula and the interosseous membrane, and forms a very thick tendon, which divides in the foot into four slips for

the four outer toes. *Flexor tibialis* is moderately developed; arising from the fibular side of the upper half of the back of the tibia, and from part of the interosseus membrane, forms a strong tendon which passes down in a groove on the back of the internal malleolus, superficial to the *tibialis posticus*, and, after entering the foot, divides into three slips,—outer, middle, and internal,—the *outer* becomes confluent with the plantar fascia, and unites with the tibial margin of the expanded *plantaris* tendon, being finally inserted into the integument of the sole of the foot along the base of the longitudinal internal callosity; the *middle* forms a long slender tendon inserted into the terminal phalanx of the rudimentary hallux; and the *internal* is inserted into the ento-cuneiform bone, and, joining the inner tendon of the *flexores breves* of the hallux, into the base of the proximal phalanx of that digit.

The middle division, forming the flexor tendon for the hallux, at first sight appears to be connected with the tendon of the *flexor fibularis*; but the connecting band is found on closer examination to proceed from the side of the os calcis across the under surface of the tendon, and seems to be the rudiment of a *flexor accessorius*.

*Thylacinus cynocephalus*.—*Flexor fibularis* is a very large and powerful muscle. It occupies the outermost place of the deep muscles on the back of the leg, and it takes origin from the whole length of the posterior surface of the fibula. The thick rope-like tendon in which it ends enters the sole by passing forwards on the back of the astragalus, and, after being joined by the tendons of the *flexor tibialis*, it splits into four, viz., a tendon for each toe. *Flexor tibialis* springs from the posterior aspects of the shaft of the tibia in its upper two-thirds, and also by a pointed

<sup>1</sup> Strangely mistaken for the *plantaris* by Owen (*Anat. Vertebr. Anim.* iii. pp. 14, 15), who also describes under the name of *flexor digitorum longus* the interosseous *pronator tibie* muscle. The true *plantaris* really arises, as usual, from the back of the external condyle of the femur, passes down under cover of the *gastrocnemius*, and its tendon, gliding over the heel bone, enters the foot, where it spreads out, becoming united with the plantar fascia, and giving off three tendons, which are inserted into the fascia and integument of the three middle toes, while from its deep surface and from the os calcis muscular fibres arise, from which four tendons are given off to form the perforated tendons of the four outer toes.

and separate process from the back of the internal tuberosity of the tibia and the posterior ligament of the knee joint. At the inner ankle it crosses the tendon of the *tibialis posticus* obliquely, and entering the sole ends by joining the large tendon of the *flexor fibularis*. *Tibialis posticus* is small in size, and intermediate in position between the *flexor fibularis* and the *flexor tibialis*. It has apparently no direct bony attachment at its origin, but springs from the fascia which covers the *popliteus*, whilst, externally, it is inseparably connected with the *flexor fibularis*. At the inner ankle its tendon passes under cover of that of the *flexor tibialis*, and proceeds along the inner margin of the foot to be inserted into the internal cuneiform bone, and into the base of the first metatarsal bone.<sup>1</sup>

*Didelphys virginianus*.—*Flexor fibularis* arises from the greater part of the back of the fibula, and forms a strong tendon, which divides into five slips for the five toes. *Flexor tibialis*, small, arises from the fibular side of the upper fifth of the tibia, lying between the *tibialis posticus* and that bone, and forms a slender tendon, which passes down superficial to that of the *tibialis posticus* on the back of the internal malleolus, and, uniting with the tendinous tibial side of the *flexor brevis* of the hallux, is with it inserted into the base of the first phalanx of that digit. Immediately after crossing the ankle joint, part of the *flexor digitorum brevis* arises from the surface of the tendon of this muscle, and, passing outwards, unites with the general mass of the muscle arising from the tendon of the *flexor fibularis*, so that, at first sight, the tendon of the *flexor tibialis* appears to be united with that of the *flexor fibularis*. *Tibialis posticus*, about the size of the preceding muscle, arises from the head of the

<sup>1</sup> Taken from Prof. D. J. Cunningham's excellent description of the anatomy of this species (*Voyage of H. M. S. "Challenger," "Zoology,"* vol. v. p. 42), the names only of the muscles having been altered. Such a remarkable difference in the mode of the attachment of the tendon of the *flexor tibialis* in two so comparatively closely allied species as *Dasyurus macrurus* and *Thylacinus cynocephalus*, has no known parallel elsewhere, as will be shown when concluding this paper, and suggests the question whether the union may not be due to the remnant of a rudimentary *flexor accessorius*, such as I have described in *Dasyurus*, having formed a connecting band between the tendons, which, originally separate in an ancestral form provided with a hallux, have, with the disappearance of that digit, become thus reunited.

fibula in close connection with the *flexor fibularis*, and forms a slender tendon, which passes down under cover of that of the *flexor tibialis* across the malleolus, but in a separate synovia sheath, and is inserted into the scaphoid bone.

In the poephagous marsupials, *Macropodidæ*, the *flexor tibialis* and *tibialis posticus* become rudimentary, or are altogether wanting. In *Macropus giganteus* the *tibialis posticus* has disappeared, and the rudimentary *flexor tibialis* arising superficially from the inner side of the head of the fibula has its very slender tendon inserted into the ento-cuneiform bone<sup>1</sup>; while in *Hypsi-prymnus gaimardi* (Plate V. fig. 9) the reduction in the number of the flexor muscles of the feet has reached its greatest extent, the *flexor fibularis* alone remaining of the three long flexors, and the short plantar muscles are altogether absent.

#### EDENTATA.

*Tamandua tetradactyla*.—*Flexor fibularis* is very large, arising from nearly the whole fibula, and forms a broad flat tendon, which in the foot receives obliquely on its plantar and inner surface the large *flexor accessorius*.

*Flexor tibialis* appears to be the direct continuation of the lower margin of the *popliteus* into the foot; its slender tendon passes downwards on the inner side of the internal malleolus in a groove by itself, and is inserted into the sesamoid bone over the ento-cuneiform bone.

*Tibialis posticus* is a small muscle arising from the heads of the tibia and fibula and intervening interosseous membrane; its tendon, which is slightly larger than that of the preceding, passes across the malleolus in a groove between those for the preceding muscles, and is inserted into the scaphoid bone.

*Cyclothurus didactylus*.—In the two-toed sloth, according to Galton,<sup>2</sup> a similar arrangement of the *flexor tibialis* (*tibialis posticus secundus*, Galton) and *tibialis posticus* exists.

*Dasypus sexcinctus*.—The myology of this species has been

<sup>1</sup> It is, of course, impossible to be absolutely sure that this muscle is not the homologue of the *tibialis posticus*, but its superficial position and mode of insertion lead me to consider it the *flexor digitorum tibialis*.

<sup>2</sup> On the Myology of *Cyclothurus didactylus*," *Ann. Mag. Nat. Hist.*, 1869.

described by Owen and Galton, and the following account of the arrangements of the long flexors has been taken from the description of these muscles by the latter writer, the names of *flexor fibularis* and *flexor tibialis* being substituted for those of *flexor longus digitorum* and *tibialis posticus* (in part) respectively.

*Flexor fibularis* arises from the whole posterior surface of the fibula, the interosseous membrane and the fibular aspect of the tibia, forms a broad tendon, which divides into five tendons for the five toes. *Flexor tibialis* arises from the middle third of the posterior surface of the tibia, immediately below the inner head of the *tibialis posticus*, with the tendon of which its tendon passes down, lying in the same groove, but to the tibial side and running over the scaphoid tubercle, is inserted into the posterior part of an ossicle which lies alongside the free edge of the ento-cuneiform bone. From the anterior margin of this ossicle a strong ligament or tendon passes, to be inserted into the tibial side of the base of the proximal phalanx of the hallux. *Tibialis posticus* arises by two short heads, the inner from the upper third of the posterior surface of the tibia, the outer from that part of the head of the fibula which is ankylosed to the tibia.

*Tolypeutes conurus*.—*Flexor fibularis* arises from the posterior surface of the fibula, the whole of the interosseous membrane, and about the middle fourth of the shaft of the tibia, and forms a strong tendon, which, passing into the sole, is there transformed into a thick solid sesamoid ossicle, from the distal end of which the five tendons for the five toes arise. *Flexor tibialis* is very small; it arises as a slight muscular slip from the inner malleolus, and is inserted into the extra free portion of bone at the proximal end of the hallux. *Tibialis posticus* has origin by a somewhat laterally compressed aponeurotic tendon from the head of the fibula, and by fleshy fibres from the back of the shaft of the tibia to the inner side and below the *popliteus*, as far as the upper end of the lower fourth of the shaft. Its tendon glides in a separate groove behind the inner malleolus, passes beneath the internal lateral ligament, and is inserted into the cuneiform bone.<sup>1</sup>

<sup>1</sup> Slightly abridged, and with the names of the muscles changed, from Dr. Murie's paper on the "Three-banded Armadillo," *Trans. Linn. Soc.*, xxx. pp. 101, 102.

*Chlamydophorus truncatus*.—*Flexor fibularis* arises and is inserted as in the preceding species. *Flexor tibialis* arises from the tibia below the insertion of the *popliteus* muscle; its tendon accompanies that of the *tibialis posticus*, and is inserted into the ossicle lying at the proximal end of the halluceal metatarsal. *Tibialis posticus* arises from the head of the fibula, in intimate union with the *flexor fibularis*; its tendon passes round the internal malleolus, and is inserted into the tubercle of the scaphoid bone.

*Orycteropus capensis*.—*Flexor fibularis* arises from the posterior surface of the fibula, from the interosseous membrane, and from the adjoining margin of the tibia, and, forming a strong flat tendon, divides into five for the five toes. *Flexor tibialis* arises in intimate union with both the *flexor fibularis* and the *tibialis posticus* from the inner aspect of the head of the fibula, the opposite-looking edge of the posterior portion of the tibia, and in part from the interosseous membrane. A little above the internal malleolus its tendon is given off, which, passing in a groove superficial to that of the *tibialis posticus*, enters the foot and divides into two slips. One joins by a broad expansion the tendon of the *flexor fibularis*, the other terminates at the posterior part of the sesamoid ossicle, which lies alongside of the ento-cuneiform bone, and which is attached by a ligament proceeding from its anterior extremity to the inner side of the base of the proximal phalanx of the hallux.<sup>1</sup>

The mode of insertion of the tendon of the *flexor tibialis*, as may be seen from the above account, differs from that of other edentates, resembling that of the Insectivore, *Solenodon cubanus* (Plate IV. fig. 2, *f. t.*). In this order, therefore, as in Insectivora, the two leading modifications of the muscle are exemplified. This adds yet another proof to the many of the correctness of Professor Flower's statement that "*Orycteropus* is a form in most respects perfectly apart from all the others."<sup>2</sup>

<sup>1</sup> Slightly abridged, and with the names of the muscles changed, from Galton's paper on the "Myology of the Upper and Lower Extremities of *Orycteropus capensis*," *Trans. Linn. Soc.* xxvi., pp. 596, 597.

<sup>2</sup> *Proc. Zool. Soc.*, 1882, p. 364.

## RODENTIA.

*Lepus cuniculus*.—*Flexor fibularis* arises from the outer surface and back of the rudimentary fibula, from the intermuscular septum separating it from the peronei muscles, from the fibular side of the head of the tibia, and from the interosseous membrane, a few fibres are attached to the fibular margin of the tibia; the muscular mass thus formed sends off two tendons—one flat and superficial, the other (the main tendon) deep and round. These tendons pass round the malleolus together. In the foot the superficial flat tendon joins the tibial side of the larger tendon (Plate VI. fig. 10), and the united tendons send off slips to the four toes, piercing the tendons of the *plantaris*. The smaller superficial muscle is evidently the rudiment of the *flexor tibialis*; the larger, the *flexor fibularis*. There is no *tibialis posticus*; a muscle which has been considered as representing it arises from the prominent postero-internal side of the head of the tibia immediately below the insertion of the tendon of the *semi-tendinosus*, forms a small muscular mass lying on the insertion of the *popliteus*, and a long tendon which passes behind the internal malleolus, and unites at the base of the first phalanx of the inner toe with the tendon of the *extensor communis digitorum* for that toe. This muscle is, however, evidently the representative of the *extensor hallucis longus*, which, in many animals possessing a hallux, sends also a slip to the *extensor communis*.

*Hystrix cristata*.—*Flexor fibularis* arises from the whole length of the fibula and from the interosseous ligament, and, forming a flattened tendon at the ankle, passes into the foot, and receives on its tibial side the smaller and similarly flattened tendon of the *flexor tibialis* (Plate VI. fig. 9), which arises from the tibia, and is connected above with the *popliteus*. *Tibialis posticus* is simple, arising from the heads of the fibula and tibia, and inserted into the scaphoid bone, its tendon passing round the malleolus with that of the *flexor digitorum*.

*Erithizon dorsatus*.—*Flexor fibularis* arises from three-fourths of the posterior surface of the fibula, and, forming a very thick tendon, passes into the foot, where it unites with that of the *flexor tibialis*; the latter arises from the tibia below the insertion of the *popliteus*, and passes round the internal malleolus in a



groove, at the bottom of which lies the tendon of the *tibialis posticus*, which is about the same size, and arises from the head of the fibula and from the adjoining interosseous membrane, and is inserted into the scaphoid bone.

*Syntheres prehensilis*.—*Flexor fibularis* is very strong, arising from almost the whole length of the tibia. *Flexor tibialis* is smaller, taking origin from half the shaft of the tibia posteriorly below the insertion of the popliteus, with which and with the *tibialis posticus* it is intimately connected; its tendon winds round the internal malleolus in a deep groove, at the bottom of which lies the tendon of the *tibialis posticus*, and, spreading out, unites with the superficial surface of the *flexor fibularis* tendon (which divides into three slips for the three inner toes), and sends a tendon to the outermost digit. *Tibialis posticus* is very large—larger than the *flexor tibialis*—arising partly under cover of it from the interosseous membrane, the adjoining edge of the tibia, and from the head of the fibula in connection with the *flexor fibularis*; its tendon, which equals that of the *flexor tibialis*, passes down with and under cover of it in the same groove on the postero-internal side of the malleolus, and is inserted into the scaphoid bone.

*Octodon cumingii*.—*Flexor fibularis* and *flexor tibialis* are both well developed; the former is the larger, arising from the fibula, the fibular margin of the tibia, and the intervening interosseous membrane. *Flexor tibialis* arises from the middle third of the posterior surface of the shaft of the tibia under the insertion of the popliteus, and forms a tendon nearly as thick as that of the *flexor fibularis*, which, passing into the foot, unites with the superficial surface of the tendon of the latter muscle (Plate VI. fig. 8). *Tibialis posticus* is quite concealed by these large muscles; it arises from the interosseous membrane and sides of the upper third of the posterior surface of the tibia and fibula, in close connection with both the tibial and fibular flexors, and forms a slender tendon, which, passing round the internal malleolus under cover of the *flexor tibialis*, is inserted into the scaphoid bone.

*Cavia flavidens*.—*Flexor fibularis* arises from almost the whole length of the fibula from the interosseous ligament, and by a few fibres from the tibia, where it is closely connected with those of

the *flexor tibialis*, and, forming a strong tendon, enters the foot, and divides into three tendons for the three toes. *Flexor tibialis* arises from the tibia below the insertion of the *popliteus*, covering and concealing the *tibialis posticus*, and forms a tendon smaller than that of the *flexor fibularis*, which it joins superficially about the middle of the sole of the foot by a flat slip; but the main tendon accompanies the inner tendon of the *flexor fibularis*, and, passing with it through the tendon of the *plantaris* (which forms the perforated tendons for the toes, as in most mammals), is inserted alongside it into the extremity of the last phalanx of the inner toe.

*Cavia cobaya*.—The arrangement of the flexor muscles and their tendons is very similar to that of *Cavia flavidens*.

*Cavia rupestris*.—*Flexor fibularis* and *flexor tibialis* arise together from the fibula, interosseous membrane, and margin of the tibia below the *popliteus*. In the lower third of the leg the slender tendon of the latter separates, and, passing across the internal malleolus with the tendon of the *tibialis posticus* (which lies on its tibial margin), enters the foot and joins the side of the large fibular flexor tendon of the toes, passing chiefly to the inner toe. *Tibialis posticus* arises by a narrow origin from the adjacent parts of the heads of the tibia and fibula, forms a moderate-sized muscle, with a larger tendon than that of the *flexor tibialis*, and is inserted into the scaphoid bone.

*Dasyprocta cristata*.—*Flexor tibialis* is comparatively small, arising from a few delicate fibres from the head of the fibula, but mainly from the back of the tibia, except the part covered by the *popliteus*. It becomes tendinous superficially above the middle of the leg, and joins in the foot the broader tendon of the *flexor fibularis*.

*Chinchilla lanigera*.—*Flexor fibularis* is large, arising from the fibula and interosseous membrane, as well as from the fascia attached to the tibia, and overlying the *flexor tibialis*; its tendon is joined in the foot (precisely as in *Potamogale velox*) by the slender tendon of the *flexor tibialis*, which arises from the upper third of the tibia under the insertion of the *popliteus*. *Tibialis posticus* has a thicker tendon, which runs alongside that of the *flexor digitorum* to the ankle, which it crosses in the same groove, but in a different synovial compartment; it arises from

the heads of the tibia and fibula, but chiefly from the latter, and is inserted into the scaphoid bone.

It is remarkable that the relations of these muscles are almost precisely similar to those of the corresponding muscles in the widely-removed *Potamogale velox*.

*Dipus ægyptius*.—The long flexor muscles are almost precisely as in *Chinchilla lanigera*; the *tibialis posticus*, however, differs in being much smaller and wholly concealed above by the *flexor tibialis*, and in having a more slender tendon (Plate VI. fig. 1). The *plantaris* forms altogether the perforated tendons for the three toes, which are but loosely connected with the bases of the first phalanges, and inserted into the sides of the distal extremities of the second phalanges. There is no trace of a *flexor digitorum brevis*, *flexor accessorius*, *lumbricales*, or *flexores breves*.

*Alectaga indica*.—In this five-toed species of *Dipodidae*, the arrangement of the long flexors in the leg is precisely similar to that of the three-toed *Dipus*. The three tendons pass downwards on the back of the tibia, touching by their contiguous margins, that of the *flexor tibialis* being between the other two. Near the ankle the tendons of the *flexor tibialis* and *tibialis posticus* are drawn slightly towards the inner malleolus, which they cross, as usual, in a groove distinct from that occupied by the *flexor fibularis*, with the tendon of which that of the *flexor tibialis* soon unites after entering the foot. The common tendon sends off slips to the first and fifth toes, and much lower down divides into three for the three middle toes. The *plantaris* supplies the perforated tendons for these toes (Plate VI. fig. 2).

*Zapus hudsonius*.—This species,<sup>1</sup> which of all the *Dipodidae* approaches the murine rodents most closely in general structure, has nevertheless also the characteristic hystricine arrangement of the flexor tendons described above. *Flexor fibularis* (Plate VI. fig. 4, *f. f.*) arises from the whole posterior surface of the rudimentary fibula, from the interosseous membrane, and from the fibular margin of the tibia under cover of the *popliteus*, and forms a strong tendon, which passes into the foot and receives on its inner side the tendon of the tibial flexor. *Flexor tibialis* (Plate VI. fig. 4, *f. t.*) is very much smaller, arising from the tibia imme-

<sup>1</sup> The type, according to Coues, of a distinct family *Zapodidae*.

diately below the insertion of the *popliteus*, soon forms a slender tendon, which passes downwards between those of the *flexor fibularis* and *tibialis posticus*, and after crossing the ankle-joint divides (as in *Solenodon cubanus*) into two (fig. 4, *a*), one unites with the tendon of the *flexor fibularis*, to form the perforating tendons for the five toes, the other goes to the base of the central callosity of the sole of the foot. *Tibialis posticus* (Plate VI. fig. 4, *t. p.*) is still smaller, arising under cover of the *popliteus* from the contiguous parts of the heads of the fibula and tibia, and from the interosseous membrane; it passes down under cover of the *flexor tibialis* to the middle of the leg, where its tendon becomes superficial, lying on the tibial side of the tendon of the *flexor tibialis*, and crossing the ankle-joint, is inserted into the scaphoid bone.

*Thomomys talpoides*.—*Flexor tibialis* arises from the tibia immediately below, and almost in continuation of, the insertion of the *popliteus*, crosses the internal malleolus in a groove internal to the *tibialis posticus*, and divides into two tendons; one goes to the sesamoid ossicle over the ento-cuneiform bone, the other is inserted into the side of the *flexor fibularis*. The latter muscle is very large, arising from the fibula and part of the tibia, and receives on its plantar surface the fibres of a large *flexor accessorius*. *Tibialis posticus* is well developed, arising between the preceding muscles from the adjacent parts of the heads of the fibula and tibia, and from the interosseous membrane between, and its tendon, passing down between those of the preceding muscles, is inserted into the scaphoid bone.

*Dipodomys phillipsi*.—The arrangement of the long flexor muscles and their tendons is as in *Dipus aegyptius* (*vide supra*), but the *tibialis posticus* is exceedingly slender, forming an almost invisible tendon.

*Bathyergus maritimus*.—In the great rodent moles of the family *Spalacidae*, on the other hand, we find an arrangement of the long flexors according to the second or *Erinaceus* type. *Flexor fibularis* (Plate VI. fig. 6, *f. f.*) is very large, arising from the whole fibula, part of the interosseous membrane, and the lower half of the tibia, and forms a strong tendon from which the five tendons for the toes are given off. *Flexor tibialis* (Plate VI. fig. 6, *f. t.*) is much

smaller, but well developed, arising from the inner side of the head of the tibia, and passing down across the insertion of the *popliteus* muscle, forms a slender tendon, which crosses the back of the malleolus, and enters the foot internal to the *tibialis posticus*, then crossing obliquely towards the internal cuneiform bone, is inserted into a broad sesamoid ossicle lying on the tibial margin of that bone, corresponding to the plantar callosity at the base of the hallux. *Tibialis posticus* (Plate VI. fig. 6, *t. p.*) is remarkably large, arising from the head of the fibula, in close connection with the *flexor fibularis*, from the adjacent margin of the tibia, and from the lower margin of the *popliteus*, and passing down in a separate groove on the malleolus, on the tibial side of the tendon of the *tibialis posticus*, is inserted into the scaphoid bone.

The *popliteus* (Plate VI. fig. 6, *p.*) is remarkable for its insertion into the head of the fibula as well as into the tibia, and the *flexor accessorius* (*f. a.*) crosses the tendon of the *flexor fibularis* in the foot, to be inserted into the base of the slip given off to the hallux.

If, with the *Dipodidæ*, we compare the anatomy of the long flexors in *Gerbillinæ*, the species of which resemble those of the former family in their long hind limbs, we find, as follows, a totally distinct arrangement.

*Gerbillus indicus*.—*Flexor fibularis* (Plate VI. fig. 3, *f. f.*) is very large, arising from the head and shaft of the fibula as far as its junction with the tibia; from the interosseous membrane, and the adjoining edge of the tibia; forms a strong tendon which divides into five tendons for the five toes. *Flexor tibialis* is altogether absent. *Tibialis posticus* (Plate VI. fig. 3, *t. p.*) is small, arising in close connection with the *popliteus*, and forming a slender tendon, which is inserted into the scaphoid bone. There is a pair of *flexores breves* for each toe, and an *adductor indicis*, in this respect also differing from *Dipus*, in which there are none; but, as in that genus, there are no *lumbricales* and no *flexor accessorius*.

The *Arvicolinæ*, on the other hand, possess the tibial flexor, but its relations are similar to those of the corresponding muscle in *Erinaceus europæus*; in the Musquash,

*Fiber zibethicus*.—*Flexor fibularis* (Plate VI. fig. 5, *f. f.*) is very

large, arising from the rudimentary fibula and interosseous membrane, and by tendinous slips from the upper half of the tibia; it forms a strong tendon, which divides into five for the five toes, receiving no *flexor accessorius*. *Flexor tibialis* (*f. t.*) is very slender, arising from the tibia as a small muscular mass below the insertion of the *popliteus*, and also from the intermuscular fascia between it and the *tibialis posticus*; it forms a slender tendon which passes down superficial and external to the tendon of the latter muscle in the same groove on the back of the malleolus, and running along the inner margin of the foot beneath the integument and areolar tissue, is inserted into the fascia covering the metatarsal bone and first phalanx of the hallux. *Tibialis posticus* (*t. p.*) arises from the tibia under cover of the *popliteus*, and above the origin of the preceding by a much thicker muscular mass, and, forming a thicker tendon, passes downwards, as above described, and is inserted into the scaphoid bone. The *plantaris* divides into four tendons for the four outer toes, those for the third and fourth divide into two slips, each of which pass forward, as usual, on either side of the perforating tendon to their insertions, while those from the second and fifth toes form only a single slip each, which, in the second digit, passes down to its insertion into the *inner* side of the middle phalanx, and in the fifth digit is inserted into the *outer* side of the corresponding phalanx. These tendons, therefore, act not only as flexors, but also as adductors of the second and fifth toes, drawing them outwards from the middle line, and so exposing the fringes of hairs (which in this species take the place of interdigital membranes), as when the animal is swimming, the hallux being abducted by the tendon of the *flexor tibialis*.

*Arvicola amphibius*.—*Flexor fibularis* as above. *Flexor tibialis* arises in close connection with the *tibialis posticus*, and appears to be a direct continuation of the *popliteus*; it forms a slender tendon, which passes down in company with that of the *tibialis posticus*, and superficial to it in the groove on the back of the internal malleolus, and, running along the internal margin of the foot, unites with the tendinous extremity of the inner division of the pair of flexores breves for the hallux, and is with it inserted into the base of the first phalanx; *tibialis posticus* arises under cover of the preceding from the tibia, and forms a slender tendon.

which, passing down as above described, is inserted into the scaphoid bone.

In *Murinae* the arrangement is generally similar to that above described; in *Sigmodon hispidus* the *flexor fibularis* is very large arising from the whole length of the rudimentary fibula, from the interosseous membrane, and from the adjoining tibial margin; *flexor tibialis* arises superficially from the head of the tibia and from the intermuscular fascia, and, forming a very slender tendon, is inserted into the tibial side of the first metatarsal. *Tibialis posticus*, from the heads of the tibia and fibula, under cover of the preceding, forms a thicker tendon inserted into the scaphoid bone.

The *Myoxidae* closely resemble the *Muridae* in the arrangement of the long flexors:—in

*Myoxus avellanarius*, the *flexor fibularis* is as in *Arvicola amphibius*; *flexor tibialis* is, however, much smaller; its very slender, thread-like tendon, after entering the foot, spreads out into a fascial aponeurosis, which is connected with the base of the central callosity of the sole, and with the sheath of the halluceal tendon from the *flexor fibularis*.

The *Sciuromorpha* closely resemble the *Myomorpha* in the anatomy of these muscles, as may be seen from the following descriptions:—

*Sciurus vulgaris*.—*Flexor fibularis* is very large, arising from the greater part of the fibula, the interosseous membrane, and by a few fibres, from the margin of the tibia; it forms a thick tendon, which receives in the foot the fibres of the *flexor accessorius*; and divides into five slips for the five toes, the four outer perforating the four tendons of the plantaris for these digits, the outermost of which receives a tendon from the *flexor accessorius*. *Flexor tibialis* arises from the greater part of the posterior margin of the tibia, some of its fibres being united with those of the preceding muscle, and forms a much smaller tendon, which passes in a distinct groove on the tibial side of the malleolus along with the tendon of the *tibialis posticus*, developing lower down a sesamoid bone, which glides over the *scapho-cuneiform* joint, and the tendon, continuing onwards over the *flexores breves* for the hallux, is, with their inner tendon, inserted into the inner side of the base of the first phalanx of that digit, precisely as in

*Sorex* (see Plate IV. fig. 4, f. t.). *Tibialis posticus* is a small muscle which arises from the heads of the fibula and tibia, under cover of the *popliteus*, and forms a slender tendon which passes downwards in a groove on the back of the internal malleolus, along with the tendon of the *flexor tibialis*, and is inserted into the scaphoid bone.

*Sciuropterus layardi*.—The arrangement of the flexors is similar to that above described, but there is only a flat ligament in place of the *flexor accessorius*; *flexor tibialis* is a very small muscle, arising high up under the margin of the insertion of the *popliteus*; its tendon runs down along with that of the *tibialis posticus*, which arises as in *Sciurus*, and both are inserted as in the preceding species; but the *flexor tibialis* is not connected by a tendinous slip with the hallux.

#### UNGULATA.

In all Ungulata, however modified the extremities may be, the arrangement of the long flexors conforms to the five-toed *Centetes* and *Solenodon* type. The following is a description of these muscles in the most specialised form, the one-toed horse, *Equus caballus*. *Flexor tibialis* is very small, arising from the postero-external surface of the head of the tibia above, and internal to the origin of the *flexor fibularis*, and between it and the edge of the *popliteus* muscle, (which occupies the whole postero-internal upper third of the tibia by its insertion); the long slender tendon passes down in a groove on the inner side of the inner malleolus, and joins the large tendon of the *flexor fibularis* about the middle of the proximal third of the metatarsal bone. *Tibialis posticus* is altogether absent.

*Tapirus sumatranus*.—*Flexor fibularis* arises from the upper three-fourths of the shaft and head of the fibula and from the interosseous membrane; its muscular belly, however, reaches to the os calcis, and covers the whole of the fibula. It there forms a strong tendon behind the os calcis, which winds round to the inner side and forms the very strong thick flattened tendon of the sole. This is joined by the *flexor tibialis* opposite the proximal end of the metatarsal, and the two unitedly passing forward about two inches, again subdivide into the three very strong thick and



broad perforating tendons of the digits. *Flexor tibialis* arises by flattened tendinous fascia from the (posterior tuberosity) styloid process of the head of the fibula, deeper and within, but in union with the *flexor fibularis*. It forms a moderate-sized belly, which crosses inwards about the middle of the tibia, and ends in a strong narrow round tendon above the malleolus. This glides in the groove behind the tibial malleolus, and crosses again somewhat outwards in a similar shallow groove, joining the broad plantar tendon of the *flexor fibularis* at the proximal end of the metatarsus. *Tibialis posticus* absent.<sup>1</sup>

*Tapirus americanus*.—In a young specimen of this species, the tendons for the three toes were found given off by the *flexor fibularis* before the *flexor tibialis* united with that muscle in the sole; each of these tendons was joined by a corresponding slip from the *flexor tibialis* before passing through the perforated tendon.<sup>2</sup> According to H. N. Turner<sup>3</sup> the *tibialis posticus* is wanting in this species also.

In these four-toed Ruminants, the arrangement of these muscles is on the same plan as in the one-toed horse, the only difference consisting in the number of tendons given off after the union of the tibial and fibular flexors in the foot; in all cases the *tibialis posticus* appears to be absent.

#### HYRACOIDEA.

*Hyrax dorsatus*.—The *flexor fibularis* is very large, arising from the fibula, interosseus membrane, and adjoining surface of the tibia. *Flexor tibialis* is smaller but well developed, from the head of the fibula with the preceding, from the interosseous membrane, and from the middle third of the tibia below, and external to the large *popliteus*; its tendon is inserted into the side of the larger tendon of the *flexor fibularis* in the foot, and sends a special slip with that of the *flexor fibularis* to the second toe. *Tibialis posticus* is altogether absent, as in *Ungulata*.<sup>4</sup>

<sup>1</sup> This account of the origin and connections of the long flexor muscles of the feet in *Tapirus sumatranus* is taken (with some necessary alterations) from Dr. Murie's paper in this *Journal*, vi. p. 165.

<sup>2</sup> Schulze, Siebold and Kolliker, *Zeitschr. Wissensch. Zool.*, 1867, ii. 15, pl. iii.

<sup>3</sup> *Proc. Zool. Soc.*, 1850, p. 106.

<sup>4</sup> The *flexor accessorius* described by Messrs. Mivart and Murie (*Proc. Zool. Soc.*, 1865, p. 350, fig. 13a) as arising from the plantar surface of the deep flexor tendon,

## PROBOSCIDEA.

*Elephas indicus*.—*Flexor tibialis* arises by tendinous and fleshy fibres from the postero-internal surface of the head of the fibula. An additional slip, small and tendinous, proceeds from the tibia, internal to the insertion of the *popliteus*, from the oblique ligament between the tibia and fibula, and from the septum between it and the *tibialis posticus*. The muscle becomes tendinous opposite the tuberosity of the os calcis, and passes into the sole along a groove at the junction of the astragalus with the sustentaculum tali. *Flexor fibularis*, a larger muscle, arises from the posterior and internal surfaces of the fibula, and from the septum between the two muscles. It passes through a distinct sheath. Opposite the ento-cuneiform bone, the *flexor tibialis*, which lies superficial to the other, spreads out, and blends by its deep surface with the tendon of the *flexor fibularis*. The separate flexor tendons are given off after this junction. The small tendon to the first digit, and that to the fifth, are furnished almost entirely by the *flexor tibialis*, while those to the middle digit proceed from the two flexors jointly (?) *Tibialis posticus* is concealed beneath the *flexor tibialis*. It arises from the posterior surface of the tibia, below what answers to the oblique line; from the adjacent surface of the fibula, and largely from the intermuscular septum between this muscle and the *flexor tibialis*. It ends in a tendon which passes through a distinct sheath on the inner malleolus, and is inserted into the upper surface of the bases of the second and third metatarsals, being crossed, immediately above its insertion, by the *tibialis anticus*.<sup>1</sup>

It appears, however, to me doubtful if this be really the homologue of the *tibialis posticus*, and I am inclined to regard it rather as the representative of the *extensor hallucis longus*, the origin of which has migrated, as in *Lepus* (*vide supra*, p. 159), to the posterior aspect of the tibia. The absence of the *tibialis posticus* would add another character uniting this order with the *Hyracoides* and *Ungulata*.

and inserted into the middle tendon of the *flexor brevis digitorum* in *H. capensis*, does not exist in Natal specimens of this species.

<sup>1</sup> Miall and Greenwood, *Jour. Anat. and Phys.*, xii. pp. 284, 285.

## CARNIVORA.

The arrangement of the long flexors in *Carnivora* may be described as being formed on the *Centetes* type (Plate IV. fig. 1). There is always a pair of long digital flexors, of which the *flexor tibialis* tendon accompanies that of the *tibialis posticus* round the internal malleolus, and is inserted into the side or superficial surface of the *flexor fibularis*. The following are descriptions of the origins, insertions, and relations of these muscles in some of the most representative species.

*Mustela vulgaris*.—*Flexor fibularis* is much larger than the *flexor tibialis*. It arises from the head and part of the shaft of the fibula and (by a large slip underlying the *tibialis posticus*) from the head of the tibia; forms a large tendon which passes into the foot, and receives the tendon of the *flexor tibialis* on its tibial side. *Flexor tibialis* arises from the greater part of the tibia under cover of, and below the insertion of the *popliteus*, with which it is closely connected, and by a slip from the head of the fibula, in connection with the fibres of the *flexor fibularis*; its tendon passes round the internal malleolus along with the tendon of the *tibialis posticus*, and is inserted into the side of the tendon of the *flexor fibularis*, the common tendon dividing into slips for the five toes. *Tibialis posticus* is well developed, arising from the heads of the fibula and tibia under cover of the *popliteus*, forms in the lower third of the leg a tendon slightly larger than those of that *flexor tibialis*, and, passing round the internal malleolus on the tibial side of the tendon of the latter muscle, is inserted into the ento-cuneiform bone. The *pronator tibiæ* is well developed and quite distinct from the *popliteus*; there is a large *flexor accessorius* which sends also a slip to the tendon for the hallux.

*Herpestes nipalensis*.—*Flexor fibularis* has a very extensive origin, from almost the whole length of the fibula, from the interosseus membrane, and from the fibular side of the tibia from about the level of the *popliteus* insertion, to within a short distance from the distal extremity of the bone; forms a thick tendon, which, passing into the foot, receives the *flexor accessorius*, and on its tibial side the tendon of the *flexor tibialis*, and divides into five slips for the five toes. *Flexor tibialis*, much

smaller, arises by a narrow slip from the head of the fibula, and by a rather extensive origin from the tibia, under cover of the insertion of the *popliteus*, and below it to a short distance below the centre of the bone, and, forming a slender tendon, which passes round the internal malleolus, and is inserted into the tibial side of the tendon of the *flexor fibularis*. *Tibialis posticus* is placed between the preceding muscles, arising from the head of the fibula, and by a few fibres from the fibular margin of the upper third of the tibia in connection with the *flexor fibularis*; it forms a slender tendon, which passes down with and on the fibular side of the tendon of the *flexor tibialis*, and is inserted into the ento-cuneiform bone.

*Viverra civetta*.—*Flexor fibularis*, fibular in origin, is quite separate from the *flexor tibialis* in the leg, the latter is tibial in origin, its tendon runs into the sole, and is there joined by that of the *flexor fibularis*, and splits into five tendons for the five toes. *Tibialis posticus* arises from the posterior surfaces of both tibia and fibula, and passes to the scaphoid bone.<sup>1</sup>

*Nasua socialis*.—*Flexor fibularis* as above. *Flexor tibialis* is comparatively large, and arises from the tibia in connection with the insertion of the *popliteus*, the *tibialis posticus* arising under cover of it from the tibia, interosseous membrane, and part of the fibula. Insertions of the three muscles as above described.

*Nandinia binotatus*.—*Flexor tibialis*, in contrast to the usual condition, is slightly longer than the *flexor fibularis*, and arises from the head of the fibula, the interosseous membrane, and the tibia under cover of the *popliteus*, with which it is more or less connected. *Tibialis posticus* is much smaller, and concealed by it, arising from the heads of the fibula and tibia, and the intervening interosseous membrane; its long flat tendon passes through the groove on the inner side of the internal malleolus under cover of the tendon of the *flexor tibialis*. The tendons of the *flexor fibularis* and *flexor tibialis* unite in the foot, and divide into five slips for the five toes; that of the *tibialis posticus* is inserted into the scaphoid bone.

*Hyena crocuta*.—*Flexor fibularis* arises from the superior extremity and upper half of the shaft of the fibula, from a corresponding extent of tibia, and from the interosseous mem-

<sup>1</sup> Young, *Jour. Anat. and Phys.*, xiv. p. 175.

brane. *Flexor tibialis* is much smaller, and springs from the head of the fibula and intermuscular septa, separating it from neighbouring muscles. The tendons of these muscles, passing through separate sheaths in the annular ligament, unite in the sole of the foot to form a single broad tendon, from which four slips are derived; these, after perforating the superficial flexor tendons, are inserted into the terminal phalanges of the toes. *Tibialis posticus* is small, and arises below the *popliteus* from the middle third of the postero-internal border of the tibia; its tendon is inserted into the scaphoid and ento-cuneiform bones.<sup>1</sup>

*Canis familiaris*.—In the dog the arrangement of the long flexors is very similar to that above described.

#### CHIROPTERA.

The arrangement of the long flexors of the feet in the bats conforms to the *Centetes* type, and resembles generally that characteristic of the insectivorous genus *Tupaia*, especially in the large size of the *flexor tibialis*, which in this order appears to reach its maximum relative development. There is but slight variability throughout the species, as may be seen from the following account of the relations of these parts in species characteristic of the leading divisions of the order.

*Pteropus samoensis*.—*Flexor tibialis* is very large, arising by a thick fleshy mass from the external condyle of the femur and from the posterior surface of the tibia, forming a strong tendon which unites in the foot superficially with the fibular flexor. *Flexor fibularis* smaller, arises from the fibular side of the head of the tibia, and from the greater part of the posterior surface of the fibula, forms a tendon smaller than that of the *flexor tibialis*, which, on entering the foot, divides into two, which again bifurcate, forming four slips, inserted *seriatim* into the tendons given off by the *flexor tibialis* for the four outer toes, the hallux being supplied altogether by the latter muscle. *Tibialis posticus* is remarkably large,<sup>2</sup> arising from the lower half of the tibia, from the interosseus membrane, and from the adjacent margin of the

<sup>1</sup> Watson and Young, *Proc. Zool. Soc.*, 1879, p. 103.

<sup>2</sup> Thus contrasting with that of *Pteropus edwardsii*, in which this muscle is described by Professor Humphry (*J. A. P.*, vol. iii. p. 13) as being small.

tibia, and forms a strong tendon inserted into the middle cuneiform bone.

*Atalpa cinerea*.—*Flexor fibularis* and *fl. tibialis* are about the same size; the former arises by a narrow head from the external condyle of the femur, and lower down where the fibula becomes strong, from the middle and part of the lower third of that bone; the latter muscle arises from the upper half of the fibular side of the back of the tibia, and, forming a longer and somewhat stronger tendon, enters the foot by passing across the internal malleolus, unites with the superficial surface of the tendon of the *flexor fibularis*, and gives off the tendons for the five toes. *Tibialis posticus* is well developed, arising between and under cover of the preceding muscles, from the interosseous membrane and the adjoining margins of the tibia and fibula, and its tendon, passing into the foot between the tendons of the above-described flexors, is inserted into the fibular side of the scaphoid bone.

The bats are thus seen to differ in the relative size, and, generally, in the relations of their long flexor muscles in the leg, from all other mammals. The *flexor fibularis*, instead of much exceeding the *fl. tibialis* in size, is scarcely equal to it, and, moreover, has its origin from the femur as well as from the fibula, except in the species of *Molossinae* (elsewhere termed by me the most quadrupedal of bats), where the fibula is well developed, and in which alone the gastrocnemius is not in an almost rudimentary condition. In this origin of the *fl. fibularis* from the femur, bats resemble birds. *Tibialis posticus*, also, differs in passing across the ankle between the two long flexor tendons, not, as in the great majority of mammals, on the tibial side of, or under cover of, the *fl. tibialis*.

*Molossus obscurus*.—*Flexor fibularis* arises from the greater part of the back of the fibula. *Flexor tibialis* arises from the heads of the tibia and fibula, but chiefly from the latter bone, and by a few fibres from the interosseus ligament, forms a tendon very nearly as large as that of the *flexor fibularis*, which it joins in the foot, and afterwards divides into tendons for the five toes. *Tibialis posticus*, from the interosseus ligament chiefly, is well developed, forming a tendon, but slightly smaller than that of the *flexor tibialis*, and is inserted into the scaphoid bone,

passing across the malleolus on the fibular side of the tendon of the *flexor tibialis*.

This exemplifies the condition of the flexor muscles in a species with well-developed fibulæ and tibiæ. In the next the fibulæ are very slender.

*Noctilio leporinus*.—*Flexor fibularis*, small, arises from the external lateral ligament of the knee joint, and from almost the whole length of the very slender fibula. *Flexor tibialis* is much larger, arising by two well-developed heads,—an external from the outer side of the head of the tibia, and an internal from the tibia below the insertion of the *popliteus*; these unite and continue to take origin from the upper half of the shaft of the tibia, and form a strong tendon, which passes down on the inner side of the malleolus, and joins the superficial surface of the tendon of the *flexor fibularis*, sending a tendon to the hallux, and uniting with the other to form the slips for the four outer toes. *Tibialis posticus* is well developed, arising from the tibia, under cover of the preceding, and from the interosseus membrane; it forms a strong tendon, which passes into the foot in a groove between the two long flexors, and is inserted into the scaphoid bone.

#### PRIMATES.

All the species conform to the *Centetes* type, although there is much variability in the manner in which the tendons for the toes are given off, often exemplified even in different individuals of the same species.

*Lemuridæ*.—*Flexor tibialis* is a very long but rather slender muscle arising from the upper two-thirds of the posterior surface of the tibia below the *popliteus* and from the peroneal side of the head of that bone, its uppermost part ascending between the tibia and the *rotator fibulæ*. It is inserted by a strong tendon which passes on the tibial side of the *flexor fibularis*, where it gives off a delicate tendon, which joins a corresponding and larger one from the *flexor fibularis* (to form the flexor tendon of the hallux), and, afterwards, becomes intimately blended with the main part of the tendon of the last-named muscle, the two giving rise to the four perforating tendons of the four outer digits, but the

*flexor tibialis* forms almost exclusively that of the fifth digit and but a small part of the others. *Flexor fibularis* is considerably larger than the preceding,—arising from the whole posterior surface of the fibula to its summit and from the whole of the interosseus membrane, also from the peroneal side of the tibia towards its distal end. In the sole of the foot it gives off a large tendon to the hallux, and then blends with the tendon of the last-named muscle.<sup>1</sup>

*Simiadae*.—The new world monkeys resemble the lemurs in the distribution of the flexor tendons. *Flexor fibularis* supplies the three middle toes, sending a slip to join the tendon from the *flexor tibialis* for the first digit. This is also the arrangement in some of the old world species, but in *Cercopithecus* and *Cynocephalus*<sup>2</sup> the hallux is supplied by a tendon from the *flexor fibularis*, which curves in a peculiar manner round the slip given off by the tibial flexor for the second toe.

*Anthropidae*.—The arrangement of the long flexor tendons in man is but a modification of that observed in *Simiadae*.

If now we tabulate the results of the above described investigation into the arrangement of the long flexor muscles of the feet in Mammalia, as follows, we shall find that they have an important bearing on the classification of the families :—

A. <i>Flexor digitorum tibialis</i> united in the foot with the <i>Flexor digitorum fibularis</i> .		B. <i>Flexor digitorum tibialis</i> not connected in the foot with the <i>Flexor digitorum fibularis</i> .	
INSECTIVORA.	<i>Chrysochloridae.</i> <i>Centetidae.</i> <i>Solenodontidae.</i> <i>Potamogalidae.</i> <i>Macrocebidæ.</i> <i>Tupaiidae.</i> <i>Galeopithecidae.</i>	INSECTIVORA.	<i>Talpidae.</i> <i>Soricidae.</i> <i>Erinaceidae.</i>
CHIROPTERA.			MONOTREMATA.
RODENTIA.	HYSTRICOMORPHA. LAGOMORPHA. <i>Orycteropodidae.</i>	RODENTIA.	MARSUPIALIA.
EDENTATA.		EDENTATA.	
HYRACOIDEA.	} <i>No tibialis posticus.</i>		
UNGULATA.			
PROBOSCIDEA.			
CARNIVORA.			
PRIMATES.			

<sup>1</sup> Murie and Mivart, *Trans. Zool. Soc.*, 1872, pp. 80, 81.

<sup>2</sup> Schulze, *l.c.*, plate ii. figs. 3, 3a.



It may be observed, firstly, that the characters derived from the two leading modes of arrangement of the long flexor tendons in the foot are applicable in a very wide sense, for, given a certain species, it may be safely assumed (as I have proved from an examination of a very large series of representative species) that every species of the family, if not of the order, to which the species in question may belong will be found on examination to possess either a similar arrangement of the flexor tendons in the feet or some slight modification not affecting the general law. Thus all the families may be found arranged under one or other of the two sections, A or B, above, and, with three exceptions only, all the orders are similarly distributed. It is further noticeable that, with these three exceptions, all the orders of Placental Mammals are referable to section A, while the Implacental Mammals fall under B. Now, as I have already shown (*vide supra*, p. 146), the arrangement of the flexor tendons in the animals included under B is a differentiated state of a primarily united condition of the homologous structures, such as we find in the species which make up the families grouped under A, it follows that the Implacental Mammals, and a few families of three orders of Placental Mammals, also, of comparatively low position, exhibit in this part of their organisation an advance in development. Furthermore, as it is difficult to conceive that in any animals in which a definite separation of the tibial from the fibular flexors had once taken place, as in those included under section B, symmetrical reunion of these tendons could subsequently occur,<sup>1</sup> so we are unable to consider that any species having the arrangement of the flexor tendons which has been described in the species representing the families and orders of that section, can represent in this respect the condition of the corresponding parts in the ancestral forms from which the great majority of Placental Mammals have sprung.

Secondly, it may be noticed that the mode of arrangement of the flexor tendons has a very important bearing in determining the natural position of certain families and groups of families. This is especially exemplified in *Rodentia* where the group *Hystricomorpha* is placed next *Lagomorpha*, and *Sciuromorpha*

<sup>1</sup> Except under the circumstances referred to above in describing these muscles in *Thylacinus* (see footnote to p. 155).

is associated with *Myomorpha*, an arrangement which, originally adopted from a consideration of other characters, has received the approval of most systematic mammalogists. The *Dipodidae*, although hitherto classed with the *Myomorpha* (by some zoologists even placed next to the true mice), must, according to the arrangement of their long flexor tendons (*vide supra*, p. 162), be removed to the group *Hystricomorpha*. That this, notwithstanding the united condition of the tibia and fibula,<sup>1</sup> is, indeed, their true natural position, is furthermore indicated, not only by the great size of the infra-orbital opening in the skull, but also by the form of the zygomatic arch and by the position of the malar (which is almost identical with that of the *Ctenodactylinae*), as well as by many other important characters. Again, although the species of *Bathyerginae* possess the so-called hystriine form of mandible, their position among *Myomorpha* is further strengthened by the characters derived from the arrangement of their long flexor tendons (*vide supra*, p. 163). In *Insectivora* the close affinity of the three families, *Erinaceidae*, *Soricidae*, and *Talpidae*, receives additional confirmation, and a single important character, not hitherto known, for separating them in a group by themselves is afforded. Finally, in *Edentata*, we obtain another most important proof of the isolated position of *Orycteropus* (*vide supra*, p. 158), which has been lately ably demonstrated by Professor Flower.<sup>2</sup>

## EXPLANATION OF PLATES IV. AND V.

### Order INSECTIVORA.

Fig. 1. *CENTETES ECAUDATUS*.—Dissection of posterior aspect of right leg and sole of foot (nat. size), showing the following muscles:—*p. popliteus*; *f. f. flexor digitorum fibularis* (= *flexor hallucis longus*); *f. t. flexor digitorum tibialis* (= *flexor digitorum longus*); *t. p. tibialis posticus*; *t. a. tendo achillis*; *pl. plantaris tendon*; *f. a. flexor accessorius*; *a*<sup>1</sup>, *a*<sup>2</sup>, *a*<sup>3</sup>, *adductor hallucis, ad. indicis, ad. minimi digiti*; underlying them the *flexores breves* may be seen; *ab.o.m. ab-*

<sup>1</sup> Far too much importance has been ascribed to the condition of the leg bones in *Rodentia* as a basis for classifying the families. The union of the fibula with the tibia in *Dipodidae*, as well as in other mammals, is a purely adaptive character, and should not be considered of more importance than, for example, the union of the metatarsals in *Dipus*, which has also evidently been brought about by special adaptation of the hinder extremities for leaping.

<sup>2</sup> *Proc. Zool. Soc.*, 1882.

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*ductor ossis metatarsi minimi digiti*; f. b. *flexor digitorum brevis*. The tendons of the long flexors of the toes are divided above the ankle, and their pedal extremities reflected, showing the distribution of the three divisions of the *flexor digitorum fibularis* (*fl. hallucis longus*) to the three middle toes, those of the *flexor digitorum tibialis* (*fl. digitorum longus*) supplying the hallux and fifth toe.

Fig. 2. SOLENODON CUBANUS.—Dissection of posterior aspect of right leg and part of the sole of the foot (nat. size). (Muscles homologous with those in fig. 1 are indicated by similar letters). The tendon of the *flexor digitorum tibialis* is seen dividing below the ankle-joint into two slips, one unites with the side of the tendon of the *fl. digitorum fibularis*, the other goes to the first metatarsal bone.

Fig. 3. ERINACEUS EUROPAEUS.—Dissection similar to that in fig. 2 (nat. size). The cut extremity of the tendon of the *flexor digitorum tibialis* is seen, which, divorced from its connection with the *flexor digitorum tibialis*, goes to the central callosity of the sole of the foot, its inner division retaining, as in *Solenodon*, its attachment to the first metatarsal.

Fig. 4. SOREX (CROCIDURA) OERULESOENS.—As above (enlarged). The plantar slip from the tendon of the *flexor digitorum tibialis* has disappeared; that for the first metatarsal bone develops a sesamoid bone, where it glides over the ento-cuneiform bone.

Fig. 5. MYOGALE PYRENAICA.—As above (slightly enlarged). *Flexor digitorum fibularis* so much increased in size that it extends upon the usual points of origin of the *fl. digitorum tibialis* and *tibialis posticus*. The latter is greatly reduced in size, and its tendon has become united above the ankle with that of the former, the united tendons sending a slip (f. t.) to the sesamoid ossicle supporting the lobular projection on the tibial margin of the foot, and then continuing onwards to its insertion into the tibial side of the first phalanx of the hallux.

Fig. 6. ERINACEUS PICTUS.—As above (enlarged). *Flexor digitorum tibialis* has altogether disappeared; the *flexor digitorum fibularis* alone forms the deep flexor of the toes.

#### Order MONOTREMATA.

Fig. 7. ECHIDNA SETOSA.—As above (reduced). *Flexor digitorum fibularis* forms the deep flexor tendons for the three inner toes; *flexor digitorum tibialis* arising, as in *Centetes*, from the head of the fibula superficial to the *fl. digitorum fibularis*, is inserted into the ungual phalanx of the hallux; *popliteus*, arising above from the external lateral ligament, is seen extending downwards to form the *pronator tibiae*.

#### Order MARSUPIALIA.

Fig. 8. BELIDEUS FLAVIVENTER.—As above (reduced). *Flexor digitorum tibialis* is seen arising from both fibula and tibia, and inserted into the sesamoid ossicle lying on the ento-cuneiform bone. *Flexor digitorum fibularis* gives off the perforating tendons for the five toes;

in the leg its superficial part (*f. p.*) forms the perforated tendons for the same digits. *Tibialis posticus*, passing down under cover of the *flexor tibialis*, sends its very slender tendon (here drawn much too thick) to the scaphoid bone.

Fig. 9. *HYPSPRYMNUS GAIMARDI*.—As above (reduced). *Flexor digitorum fibularis* alone remains, the other two deep flexors having altogether disappeared, as well as the plantar flexor and adductors, illustrating the extreme degree of reduction in the number of these muscles.

## EXPLANATION OF PLATE VI.

### Order RODENTIA.

Fig. 1. *DIPUS ÆGYPTIUS*.—(Muscles, homologous with those referred to above in the corresponding parts of *Centetes ecaudatus*, are indicated by the same letters). Dissection as above (nat. size), showing arrangement and mode of union of the tendons of the *flexor digitorum fibularis* and *flexor digitorum tibialis*, and position and insertion of that of the *tibialis posticus*.

Fig. 2. *ALLECTAGA INDICA*.—As above (nat. size), except that the whole foot is shown in outline, with the divided and reflected tendon of the plantaris (*pl'*), which gives off the perforated tendons for the three middle toes.

Fig. 3. *GERBILLUS INDICUS*.—As fig. 1 (nat. size). The *flexor digitorum fibularis* has altogether disappeared.

Fig. 4. *ZAPUS HUDSONIUS*.—As fig. 1 (slightly enlarged). The tendon of the *flexor digitorum tibialis*, before uniting with that of the *flexor digitorum fibularis*, sends a slip (*f. t'*, better shown in fig. 4 a, enlarged), to the base of the central callosity of the sole of the foot.

Fig. 5. *FIBER ZIBETHICUS*.—As fig. 1. The tendon of the *flexor digitorum tibialis* has lost its connection with that of the *fl. digitorum fibularis*, but the representative of the plantar slip in *Zapus* (*f. t'*) remains.

Fig. 6. *BATHYERGUS MARITIMUS*.—As above. *Flexor digitorum fibularis* and *tibialis posticus* are so large that the *flexor digitorum tibialis* has changed its origin (as in *Solenodon cubanus* and *Erinaceus europæus*, Plate IV. figs. 2 and 3) to the inner margin of the head of the tibia; in the foot the relations are similar to those in fig. 5.

Fig. 7. *CAVIA FLAVIDEUS*.—Enlarged drawing, showing mode of union of the long flexor tendons in the foot, also the separate tendons given by each of these muscles to the inner toe.

Fig. 8. *OCTODON CUMINGII*.—Enlarged drawing, showing mode of union of the long flexor tendons in the foot, and junction of the *flexor accessorius* (*f. a.*).

Fig. 9. *HYSTRIX CRISTATA*.—Drawing, (reduced), of long flexor tendons, showing their modes of union in the foot, and division into five slips, (with *lunbricales*), for the five toes.

Fig. 10. *LEPUS CUNICULUS*.—As above.

OBLITERATIVE ENDARTERITIS, AND THE INFLAMMATORY CHANGES IN THE COATS OF THE SMALL VESSELS. By ROBERT SAUNDBY, M.D. Edin., M.R.C.P. Lond. (PLATE VII.)

INFLAMMATORY changes in the smaller vessels have been comparatively little studied in this country. Several of these appearances have never been published in any English work, while some are now described for the first time.

German histologists, largely influenced by the opinion expressed by Virchow<sup>1</sup> that the inner coat of the vessel does not participate in inflammatory changes, were for long blind to these alterations. Rindfleisch,<sup>2</sup> in the second German edition of his work, declares that the inner coat seldom undergoes much alteration.

In France, MM. Cornil and Ranvier<sup>3</sup> recognised that the small vessels embedded in inflamed tissues shared in their inflammation, and that endarteritis and obliteration of the lumen were frequent occurrences in these conditions.

But probably the term obliterative endarteritis became first generally known in this country on the publication of Friedländer's<sup>4</sup> paper. He pointed out that it occurs in simple granulation tissue, gummata, tuberculosis with commencing chronic inflammatory or ulcerative processes; more rarely in sarcomata. In fibromata, and especially in elephantiasis, it is very distinct; also in cancer, where the connective tissue elements are strongly developed.

In the present paper I intend to describe and figure some of the common types, including especially the appearances in gummata and Bright's disease.

If one may be allowed to generalise at the outset, I should

<sup>1</sup> "Ueber d. akute Entzündung der Arterien," *Virchow's Archiv*, vol. i. p. 272.

<sup>2</sup> *Traité d'histologie Pathologique* (French Trans.), Second German edition, 1873, p. 222.

<sup>3</sup> *Manuel d'histologie Pathologique*, 1869-73, p. 555.

<sup>4</sup> *Contrib. f. d. Med. Wissenschaft.*, January 22, 1876.

say that the changes in the vessels agree very closely in character with the changes in the surrounding tissues. Where the inflamed parts are swollen and infiltrated, swelling and infiltration will be found in the vessels; and where the inflammation gives way to organisation, however low in type, this is repeated in the vascular walls.

In a paper on the "Process of Healing," Dr. D. J. Hamilton,<sup>1</sup> now of Aberdeen, has insisted upon the importance of simple swelling, or œdema, in the formation of granulation tissue. Often the main histological difference between an inflamed structure and one in health depends upon this condition of succulence or œdema. By it the cells are separated, their nuclei become visible, and take on vivid staining with colouring agents.

This is the change called by French histologists "embryonic transformation." In granulation tissue we may consequently expect that any vessels embedded in it would present such appearances as naturally result from swelling of their coats, with or without any alterations in structure due to the addition of new elements. The simplest condition of *swelling* of the coats is illustrated in fig. 1, a section of a small artery from the wall of an acute abscess. The section is not made quite at right angles to the axis of the vessel. The muscularis is much swollen, so that the nuclei of its muscular fibres are very visible.

Fig. 2 is a small vessel from old *granulation* tissue, obtained from the orifice of a sinus in a case of strumous disease of the hip. The lumen is nearly obliterated by the swollen and proliferating endothelium. The remainder of the wall is distended with œdema, so as to separate the individual elements, while there are numerous lymphoid cells scattered through it, or collected in groups at the periphery. The vessel depicted is a well-marked example; but similar appearances are to be seen without difficulty, especially if rather old granulation tissue be selected.

When the inflammatory process is more *chronic* and associated with new formation of tissue, the changes in the vessels present corresponding characters. Fig. 3 is a section of a pulmonary vessel from a case of phthisis. The lumen of the vessel is

<sup>1</sup> *Journal of Anatomy and Physiology*, vol. xiii.

almost obliterated by a growth of low organisation, containing a few spindle and round cell elements. The *muscularis* is apparently atrophied; the position of the remains of the *elastic lamina* is marked at *a*.

The obliteration of vessels in *tuberculosis* is alluded to by Cornil and Ranvier, but they imagine that it is effected by pressure on the vessel causing slowing of the blood-stream, and ultimately thrombosis, the thrombus becoming organised later on. If this were so in the present case the thrombus must have been parietal, as the lumen remains free;<sup>1</sup> but it is now known that the so-called organisation of thrombus is in reality an obliterative endarteritis, in which the thrombus plays merely a passive part. It is certain that the formation of a thrombus is not essential, and, in the absence of blood pigment or other evidence of disintegrated clot, it seems quite superfluous to assume that it has intervened in the process.

When the inflammatory products are *fibroid* in character we have such appearances as are seen in fig. 4 from the ovary of an adult. The *intima* is enormously increased in thickness, so as to diminish the lumen very greatly, and is lined by a layer of endothelium; the *muscularis* seems atrophied.

These appearances have been described by Noeggerath<sup>2</sup> and Patenko;<sup>3</sup> by the former they were regarded as playing an important part in the genesis of ovarian cysts; but, as Patenko has shown, they are always to be found in the neighbourhood of the small fibroid patches, which result from the cicatrization of ruptured follicles, or the imperfect development of others, and are simply examples of endarteritis, in which the vessels share in the fibroid character of the organisation which has taken place.

This condition appears to me to be anatomically identical with that described by Sir William Gull and Dr. Sutton<sup>4</sup> as *arterio-capillary fibrosis*, in connection with Bright's disease.

As is well known, they described an affection of the smaller

<sup>1</sup> I am aware that the lumen is said to be sometimes restored when thrombosis has been complete.

<sup>2</sup> *American Journal of Obstetrics*, vol. xiii. p. 1.

<sup>3</sup> *Virchow's Archiv*, vol. lxxxiv., 1881.

<sup>4</sup> *Médec.-Chir. Trans.*, vol. lv. p. 273; *Trans. Path. Soc.*, vol. xxviii. 1877.

arteries and capillaries, consisting of a deposit of a hyaline-fibroid material in the outer and inner coats, causing atrophy of the *muscularis*. They regard this vascular lesion as a primary disease, leading to various nutritive derangements in the tissues around. Their descriptions were taken from the kidney in the contracting form of Bright's disease, and are supported by the appearances to be seen in that condition; but I have never been able to confirm their statement, that a similar change may be seen in the vessels of the pia mater, mesentery, &c. In these I have been able to see only thickening of the *muscularis*, with, in some cases, some thickening of the *adventitia*, but never any growth from the intima. Ewald<sup>1</sup> and Senator<sup>2</sup> both take the view that arterio-capillary fibrosis is simply secondary endarteritis. In their second paper Gull and Sutton do their best to support their original proposition; but the vessels figured are so small that it is impossible to discover what parts of the wall are thickened; while in many instances there are local changes present, which would fully account for the vascular lesions, as in plate xxix., which represents a section of the cord, from an old case of syphilitic paraplegia.

They have never attempted to describe minutely the appearances of a vessel affected with arterio-capillary fibrosis. This omission I have endeavoured to supply in fig. 5, a drawing of a portion of a vessel from a typical case of contracting kidney. The *elastic lamina* (*a*) is much swollen, and its layers are separated and infiltrated. To the inner side is a broad band of hyaline tissue, containing a number of spindle cell elements and some elastic fibres. On the outer side is the *muscularis*, certainly not hypertrophied, but cedematous; its individual muscular fibres are separated, and their nuclei stain distinctly. The *adventitia* is blended intimately with the surrounding tissue, so that its limits cannot be defined.

If all the vessels of the contracting kidney were like this, Gull and Sutton's description would in the main be true, but many differ very considerably, and conform much more to Johnson's notion of thickening from muscular hypertrophy. Fig. 6 is a drawing of a section of a vessel of the same size, and from the same kidney as the last. It seems to be chiefly made up of

<sup>1</sup> Virchow's *Archiv*, Bd. lxxi. Hft. 4.

<sup>2</sup> *Ibid.*, Bd. lxxii. Hft. 1.



muscular fibres. It is impossible to withhold the admission that we have to do here with a numerical hyperplasia of the transverse muscular fibres. There is a pretty general agreement that this actually does take place, Ewald<sup>1</sup> has definitely admitted it, and although Cornil and Ranvier<sup>2</sup> deny it, I think its occurrence must be regarded as undoubted. In a section of kidney many more vessels or sections of vessels may be seen conforming to this second type, and it seems reasonable to believe that the hypertrophy is the general lesion affecting all the vessels and due to systemic causes, while endarteritis is the necessary accompaniment of the inflammatory processes going on in the kidney, and which affects the vessels in patches or particular parts of their course on account of the inflammatory process in the neighbourhood being particularly active.

The subject of *syphilitic disease* of the vessels is a little complicated. In the first place, there are the descriptions of vessels which were probably not syphilitic at all, but simply examples of ordinary obliterative arteritis, such as those in the so-called syphilitic placenta,<sup>3</sup> and in a case of syphilitic insanity.<sup>4</sup>

Mr. Lawson Tait<sup>5</sup> has drawn attention to the resemblance of the vessels in syphilitic placenta to those met with in the contracting kidney, and he points out that there are no appearances in such placentaë which can be regarded as characteristic of syphilitic or gummatous infiltration. In conjunction with Mr. Howard Lowe, I undertook some investigations into this question, the results of which were published in the *Ingleby Lectures* for 1879,<sup>6</sup> delivered by Dr. Bassett, and I quite agree with the statement just quoted from Mr. Lawson Tait.

The case of syphilitic insanity was published before the appearances of simple endarteritis were generally recognised, and I believe the author himself no longer maintains the specific nature of the lesions he described.

In the second place, we have the alleged occurrence of primary disease of the cerebral arteries, the histological

<sup>1</sup> *Loc. cit.*

<sup>2</sup> *Op. cit.*, p. 1064.

<sup>3</sup> *Nord. Med. Ark.*, I. 4, H. p. 73.

<sup>4</sup> *Journal of Mental Science*, Oct. 1874.

<sup>5</sup> *Obstet. Soc. Trans.*, vol. xvii.

<sup>6</sup> *Birmingham Med. Review*, 1879, p. 378.

details of which were first elaborated by Heubner.<sup>1</sup> He maintains that the affection commences in the intima, and is essentially a gummatous growth starting from thence. On the other hand, Friedländer,<sup>2</sup> Baumgarten,<sup>3</sup> and others hold that it is a non-specific endarteritis, the sequel to gummatous infiltration of the adventitia. It is not pretended by either side that the endarterial lesion presents any characters by which its specific nature can be positively identified, and the whole dispute turns on the seat of the initial lesion. I have never seen a case of arterial syphilis in which there was not marked infiltration of the adventitia, nor do I know of any case in which this has not been present. I am therefore inclined to agree with Baumgarten and Friedländer in regarding it as an endarteritis dependent upon the gummatous growth, and I shall not attempt to give it a separate description.

In the third place there are the changes in the vessels embedded in, or near to, gummata. The tendency of syphilis to invade the sheaths of the vessels has been long observed, and vessels in the neighbourhood of gummata undoubtedly present more general and more extensive changes than are to be met with in connection with any other neoplasm.

Fig. 7 is a section of a small vessel from an old brain gumma, the greater part of which was in a state of caseation; this vessel was in a recent and active looking part of the growth, which stained well and was quite translucent. The vessel has in part broken away from its surroundings. The coats are formed of concentrically arranged fibres strewn with lymphoid cells. The endothelial lining has disappeared. To the left at *a*, is another much smaller vessel with thickened walls. The analogy between this and fig. 2 cannot be overlooked, and whatever theory may be employed to explain the appearances in one, must be equally applicable to the other. The only difference in the two vessels appears to be that the lesion in fig. 7 is of somewhat older standing, and the disappearance of the normal elements of the wall is greater.

The changes in a vessel of larger size in consequence of gum-

<sup>1</sup> *Dieluetische Erkrankung der Hirnarterien*, Leipzig, 1874.

<sup>2</sup> *Op. cit.*

<sup>3</sup> *Virchow's Archiv*, Bd. lxx. H. 1.

matous infiltration are seen in fig. 8, which is a drawing of a section of a small artery of the pia mater from a case of syphilitic hemiplegia in a young subject. The bright line of the *elastic lamina* at *a*, which is not very well brought out in the drawing, serves as a landmark. On the inner side of that is the broad layer filling up half the lumen, and composed of embryonic connective tissue. Outside *a* is the *muscularis*, possibly atrophied, certainly infiltrated and surrounded by gummatous growth. In the obliterating growth at *b* is what appears to be a giant cell.

According to Heubner, the endarterial growth undergoes incomplete organisation, imitating the structure of the normal arterial coats, and being to some extent differentiated into an *intima*, composed of embryonic connective tissue and a *muscularis* formed of large spindle cells running transversely, the two layers being divided by a brightly refracting line of elastic tissue representing the *elastic lamina*. The newly formed imperfect elastic layer can be easily seen in many specimens in which the growth has become fairly organised, but it is not visible in the present specimen, and the whole description, though apparently somewhat fanciful, is fairly borne out by the microscopical appearances.<sup>1</sup>

#### DESCRIPTION OF PLATE VII

Fig. 1. Hartnack, Oc. 3, Obj. 4. Tube drawn out. Section of swollen arteriole from wall of abscess.

Fig. 2. Hartnack, Oc. 3, Obj. 4. Tube drawn out. Section of small vessel from granulation tissue; *a*, swollen and proliferating endothelium; *b*, swollen transverse muscular fibres; *c*, groups of lymphoid cells in adventitia.

Fig. 3. Hartnack, Oc. 3, Obj. 4. Tube drawn out. Section of vessel from phthisical lung, showing obliterative endarteritis; *a*, elastic lamina.

Fig. 4. Hartnack, Oc. 3, Obj. 4. Tube drawn out. Section of sclerosed artery from ovary; *a*, elastic lamina.

Fig. 5. Hartnack, Oc. 3, Obj. 8. Tube drawn out. Part of section of vessel from contracting kidney, showing obliterative endar-

<sup>1</sup> Dr. Greenfield has given a very good description of these alterations in the twenty-eighth volume of the *Pathological Society's Transactions*.

teritis; *a*, elastic lamina, swollen and infiltrated; *b*, obliterating growth surrounding lumen; *c*, muscularis oedematous.

Fig. 6. Hartnack, Oc. 3, Obj. 8. Tube drawn out. Part of section of vessel from contracting kidney, showing muscular hyperplasia.

Fig. 7. Hartnack, Oc. 3, Obj. 4. Tube drawn out. Vessel from margin of cerebral gumma; *a*, a smaller vessel with thickened wall.

Fig. 8. Hartnack, Oc. 3, Obj. 4. Tube drawn out. Vessel from gummatous infiltration of pia mater at base of brain; *a*, elastic lamina.

THE PRESENCE OF A TYMPANUM IN THE GENUS  
RAIA. By GEO. BOND HOWES, *Demonstrator of Biology,*  
*Normal School of Science, S. Kensington.* (PLATE VIII.)

HAVING recently had occasion to look carefully into the anatomy of the auditory organ of the common Skate, I have been struck with the relations of the structure detailed below, and known as the "fenestra vestibuli cartilaginei." Although long ago described, its real significance does not appear to have been shewn. Further, it is usually stated that the auditory organ of plagiostomes is completely enclosed in cartilage, Chimaera alone being exceptional among Elasmobranchs.

If the auditory capsule of the skate be laid open from above, there will be seen in its postero-internal region a recess, fig. 2, *Ty*, the area of which is roofed in by a membranous elastic fenestra, the cartilage having been absorbed. It lies posterior, and slightly internal to the opening of the aqueductus vestibuli, figs. 1 and 2, *v. aq.*, and its relatively immense size in the genus Raia, at once strikes one, on examining the macerated skull. See fig. 1.

Monro, in his classical work cited below,<sup>1</sup> first accurately described the skate's auditory organ. Making no mention of this structure, he explained away the absence of "the membrane of the drum and cavity of the tympanum" and their associated parts, by arguments untenable in the present state of physical science. Later, Müller described<sup>2</sup> a hollow or depression (the "parietal grube" of Gegenbaur<sup>3</sup>) in the middle of the occipital region of the skull, which is "covered by skin, and receives four openings," obviously referring to those of the aqueductus vestibuli, and the structure now under consideration. Gegenbaur (p. 49) describes them as leading into the labyrinth, but his figure does not do justice to the skate, as regards the hinder of the two. Scarpa<sup>4</sup> first described this, and likened it to the fenestra

<sup>1</sup> *On the Structure and Physiology of Fishes, &c.*, Edinburgh, 1785.

<sup>2</sup> *Elem. of Physiology*, English translation, 1837.

<sup>3</sup> *Das Kopfskelet der Selachier*, Leipzig, 1872.

<sup>4</sup> *Anat. disquisitio de auditu et olfactu*. Ticini, 1789.

of the higher forms. Comparison of his figures and description seems to show that he confused it with the *saccus endolymphaticus*, with which it has nothing to do; and in the most recent researches upon the Elasmobranch's auditory organ,<sup>1</sup> Retzius speaks of *ovalis* as comparable to the *fenestra rotunda*, figuring it in his elaborate treatise lately published.

If the skate be compared with certain other members of the group, it will be seen that the conditions met with respecting this structure are correlated with the flattening which that animal has undergone. In the laterally compressed forms, such as *Scyllium*, *Cestracion*, and *Chimaera*, in which the muscles of the back are prolonged well up on to the hinder region of the skull, it is either absent or small. In the Monkfish (*Squatina*) it is present, though relatively smaller than in the skate, and lies at the base of the parietal fossa above mentioned; as in *Scyllium* the skin stretches across this, and there is a mass of subcutaneous tissue interposed between the two. The flattening and lateral extension and their accompanying changes, seen commencing in *Squatina*, reach their maximum in *Raia*, the result being that the integument fits more closely into the recesses of the now almost obliterated parietal fossa; and as the muscles are relatively less developed here, there is established a closer proximity between the skin *a.* and the underlying fenestra, which thereupon becomes enlarged, forming a "tympanium," *c.* fig. 3. There is no union of adjacent parts with marked thinning of the integument, such as characterises the tympanic membrane of the higher forms, but the skin, elsewhere firmly adherent to the body, has at this point interposed between it and the membranous tympanium, a homogenous semi-fluid layer, fig. 36, such as favour the transmission of the sound wave, *b.*

It is obvious that any tendency to fenestration of an exposed surface of such a capsule must necessarily render the auditory organ so much the more efficient, and in view of the elaborate accessory structures related to the Teleostan apparatus, the above considerations render it interesting to observe that there exists in the generalised Elasmobranch this indication of the essential

<sup>1</sup> "Zur Kenntniss v. d. Membranösen gehörlabyrinth b. d. Kuorpelfischen." *Arch. f. Anat. and Phys.*, 1878; *Das Gehörorgan der Wirbelth.*, vol. i., Stockholm, 1881.

process involved in the auditory organ of the higher forms. The resulting structure, in a sense comparable to one of the fenestrae of the amniote, anticipates physiologically the initial step in the elaboration of the auditory organ in the ascending series of vertebrata.

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#### EXPLANATION OF PLATE VIII

Fig. 1. The posterior half of the macerated skull, seen from above.

Fig. 2. The auditory capsule of the right side, seen from within, the inner half removed.

Fig. 3. The same, cut to the level of the tympanum.

Fig. 1 reduced one-half ; Figs. 2 and 3 natural size.

THE LIGAMENTUM TERES. By J. B. SUTTON, *Demonstrator of Anatomy, Middlesex Hospital.* (PLATE VIII.)

THE *round ligament* of the coxo-femoral articulation has long been an anatomical puzzle, consequently many diverse notions exist concerning it. Some anatomists ascribe to this hollow band of fibrous tissue very important functions in the mechanism of the hip-joint; others deprive it of all glory, simply assigning to it the menial duty of conveying blood-vessels to the head of the femur.

Certain authors content themselves with a brief description of its attachments; others give elaborate and detailed accounts, extending over several pages, of its anatomy and supposed function. My object in the present communication is to show that this singular ligament is nothing more than the tendon of the pectineus muscle, separated from it in consequence of skeletal modifications.

Careful attention to the mode of development of the ligamentous bonds of diarthrodial articulations has led me to formulate two rules with regard to them—viz.,

1. Many ligaments arise as thickenings in a capsule, due to prolonged tension in definite directions, the capsular ligament itself being merely the perichondrium passing from one cartilage to another.

In this manner distinct bands of tissue form on the sides, and other aspects of joints which afterwards differentiate into distinct ligaments.

2. Many ligaments are the *tendons of muscles* which were originally in relation with the joint; but the parent muscle has either formed new attachments or become obsolete, whilst its tendon remains as a passive element in the articulation.

To this group belongs the *lig. teres*, a structure fairly constant throughout Mammalia, though absent in the elephant, seal, &c. (sometimes wanting in man, says Meckel).

It is almost universal among birds; indeed, among these highly specialised and beautiful creatures it reaches a high standard of development.



The attachments and relations of the ligament in man are so well known that description is needless here, merely noting that in the foetus at birth it is very strong and dense.

In the horse one gets the first glimpse of its true nature.

In this animal the ligament consists of two bands—one hidden within the joint, termed the cotyloid portion; the other passes out of the cavity to join the linea alba at its junction with the pubes, hence termed the pubio femoral portion. From this band the pectineus takes part of its origin.

These facts led me to investigate the ligament in the ostrich, holding as it does a corresponding position among birds as the horse does among mammals, for cursorial powers.

In the ostrich (*Struthio camelus*) the ligamentum teres has a true tendinous structure. It is dense and strong, contains a large quantity of elastic tissue, and has its individual fasciculi arranged regularly, corresponding to the arrangement encountered in the rounded tendons of limb muscles. In my specimen the ligament was three-fifths of an inch in thickness.

On making a section horizontally through the acetabulum, so as to include the ambiens muscle at its origin, and the ligament (see fig. 1), it may be clearly seen that they are continuous with one another across the cotyloid cavity by fibrous tissue.

The ambiens is a muscle of considerable interest, partly on account of its variability, but also on account of its curious course and relations.

It is fusiform, and arises from the tip of the short, anteriorly directed spine, which is situated above the anterior border of the acetabulum, and runs along the inner border of the thigh to the knee, where it is covered by the sartorius. Here it degenerates into a round and polished tendon, crossing in front of the knee, running in the substance of the fascia of the extensor tendon, immediately in front of the patella, to the outer side, where it joins the fibres of origin of the *flexor perforatus digitorum*.

Remembering that birds and mammals had a common origin, one turned for farther information to that curious lizard, *Sphenodon*. In this remarkable creature the hip-joint is of simple character, and the muscle corresponding to the ambiens of birds and the pectineus of man arises by two heads—one from the lateral spine of the pubes; the other is reflected from the muscle to the

inside of the capsule, so as to gain an attachment to the head of the femur; thus holding a corresponding relation to the joint as does the ligamentum teres of man and birds (see fig. 2).

The varying relations of muscle and ligament may be arranged in a tabular form, thus :—

1. *Sphenodon*.—Tendon of ambiens (pectineus) passes inside the capsule to the head of femur.
2. *Struthio*.—Lig. teres continuous with ambiens (pect.) across the cotyloid cavity by fibrous tissue.
3. *Equus*.—Lig. teres in two parts—one continuous with pectineus outside the joint.
4. *Homo*.—Lig. teres a fibrous band, carrying blood-vessels to the head of femur.

Besides the direct evidence, other facts may be mentioned respecting the ambiens to show that it is a muscle of great variability.

In comparing it with pectineus, it would seem that the crural portion alone persists in mammals.

Its crural and lower parts in birds; but all parts, upper, crural, and lower—luxuriate in full perfection among lizards and crocodiles.

For more detailed accounts of this peculiar muscle, the reader must refer to papers by Garrod, published in the *Proc. of Zoolog. Society*, 1873, "On certain Muscles of the Thigh in Birds," and an extremely valuable paper by Dr. Hans Gadow, in *Journal of Anatomy and Physiology* for July 1882.

## FIBRINOUS COAGULA IN THE LEFT VENTRICLE.

By ALEXANDER M. M'ALDOWIE, M.D., Stoke-upon-Trent.  
(PLATE IX.)

FROM time to time cases occur in practice, in which patients, whose strength has been greatly reduced by some chronic disease, die suddenly and somewhat unexpectedly, the death coming as a severe shock to the friends. In a certain proportion of these cases, firm white coagula are found in one or other of the cavities of the heart. In cases, too, of slow death, where the patient has been almost moribund for many hours or even days, the end comes suddenly whilst the patient is being raised up in bed, or on making some slight exertion. In many of these cases fibrinous clots are also found. These have long been known to have been formed anterior to death, but the probability of their being the immediate cause of death has only been fully recognised since Richardson published his researches on the different conditions of the blood in the heart after death.

Cardiac clots are divided into two classes; the static or dark clot, formed after death, and the dynamic or fibrinous clot, deposited while the blood is in circulation. The latter is subdivided into two varieties, viz.:—those organically adherent to the walls of the heart, and those whose attachment is merely mechanical. It is the last variety of clot alone, which will be referred to in the present paper.

The prevailing idea is that this form of deposition occurs much more frequently in the right cavities than in the left, but in twenty consecutive cases of ante-mortem concretion which I examined, the coagula were found on both sides of the heart in fifteen instances, in the right cavities alone in two cases, and in the left alone in three. In most instances, however, the clots were much smaller in the left than in the right chamber. They may, therefore, frequently be overlooked in superficial examinations of the heart. There is this further difference between the sides. In the right the favourite site of deposition is the auricle, the clot being rarely found in the ventricle alone, while in the left side the ventricle is the most frequent seat. When

deposition takes place in both sides of the heart all the cavities usually contain coagula, but when the left side is alone affected, then the auricle may be filled with post-mortem clot or with fluid blood.

In nearly all the cases which I have investigated the deposition of fibrin has begun at the anterior segment of the mitral valve. The clot grows at first upwards towards the aortic orifice, and is adherent by its base only, the adhesion being mechanical and produced by an interlacing of the fibrin with the chordæ tendineæ. It is quite thin and flat, but when it reaches the aortic orifice it becomes very much constricted by the approximation of the sides of the orifice during the contraction of the ventricle. These clots always exhibit this constriction to a very great extent no matter what size they attain, and it is one of the chief marks of distinction between them and those which are formed in the right ventricle. These latter often show no constriction opposite the opening of the pulmonary artery, occasionally they do, but never so markedly as in left ventricular concretions. As the deposition proceeds the clot extends along the interior of the aorta, where its form becomes modified by the altered physical conditions to which it is subject. The aorta is always more or less dilated, therefore this portion of the clot is not subjected to intermittent compression like that which lies within the heart. The clot, therefore, usually shows a very decided bulging just above the mouth of the aorta, and from this gradually tapers away till it terminates in a fine point. It is at first flat and ribboned-like, but as deposition proceeds it becomes rounded in form. In some cases the aortic portion is only two or three inches long, in others it extends for ten or twelve inches along the interior of the vessel, sending filaments into the branches which rise from the arch. The clot at the junction between the aortic and the ventricular portions is sometimes only a mere filament, so that it appears wonderful that the large mass in the aorta had not been washed away by the force of the circulation.

In addition to the constriction the coagula usually show three indentations at the points where the corpora Arantii of the semilunar valves strike in their recoil. These markings are not so regular as we would expect when we consider the purely

mechanical nature of their production. Often there are but two indentations, occasionally I have observed only one, the other side being smooth and flat. In most of the cases I observed that the left posterior valve caused a deeper depression than the others. These valvular impressions are also usually observed in clots which form in the right ventricle, but their appearance is somewhat different from those at present under notice (see fig. 2, Plate IX.).

In some instances the portion of the coagulum within the heart is small, and only occupies that part of the ventricle between the mitral and the aortic orifices. In fig. 2, Plate IX. for instance, the aortic clot was 10 inches long, and branched into the innominate, left carotid, and subclavian arteries, whilst the ventricular portion was a thin flat triangular coagulum only  $\frac{7}{8}$  inch in extent. Usually, however, this part of the concretion enlarges in the manner which I shall now describe. In its early stage the clot floats loosely in the cavity, moving up and down with the changes in the direction of the blood current, the fibrin being deposited on its surface both during the auricular and the ventricular systole; but afterwards, when it is prolonged into the aorta, its position becomes fixed, and deposition takes place only during the systole of the ventricle. The clot then begins to grow towards the apex of the heart by sending out filaments, which attach themselves to the columnæ carneæ along the anterior and left sides of the cavity. The spaces between these filaments are gradually filled up as the deposition proceeds. If the auricle also should contain a concretion, it is prolonged through the auriculo-ventricular orifice and attached to this portion of the ventricular clot. Frequently deposition takes place near the apex at the same time or soon after it occurs at the mitral valve. The fibrin is deposited in the form of fine filaments adhered to the columnæ carneæ, but these soon become fused with the larger clot. The surface of the concretion, which is directed towards the interior of the cavity, is smooth and more or less flattened; but if life be sufficiently prolonged, it bulges into the chamber and becomes channelled or otherwise marked by blood currents. One or two concretions presented an appearance as if two clots had coalesced, but I think that this was only surface markings produced by the action of the blood.

The clots may afterwards be moulded by the circulation into many forms, but they generally retain more or less the main features which I have described, which show their origin and growth.

Between the points where the fibrinous concretion are adherent to the ventricular wall, small dark blood clots are frequently found. Many authorities look upon these as being the cause of the deposition of fibrin. They allege that when the heart is much dilated the recesses between the columnæ carnæ do not entirely close in the systole, but remain as permanently patulous cavities, in which blood, if the general current is very slow, may be entirely motionless, and so be ready to coagulate. Once formed, the surface of the clot receives additions of fibrin, layer by layer, as any other unnatural surface in the blood stream always does.<sup>1</sup>

There is a form of concretion which originates in this manner, but it is formed at a later period, is soft and friable, and is usually found in the right cavities of the heart. In the variety which I have described, coagulation of the blood takes place after the deposition of fibrin between the digitations which spread along the walls of the ventricle during the growth of the clot. Richardson states that in fibrinous concretion the first step in development consists in the laying down of a portion of fibrin, in such a manner as to form a fixed point or basis for future deposition.

These fibrinous clots are mostly found in cases of slow death, and the prevailing opinion at the present time is that they are caused by stagnation of the blood current. In many cases of slow death, however, they are absent, the heart containing only fluid blood, or dark amorphous blood-clot. And the fact appears to have been overlooked that they generally take their origin at the point where the circulation is most active, and the blood constantly in motion, viz., the segment of the mitral valve which lies between the mitral and aortic orifices of the ventricle. It is more probable that the deposition is entirely due to some change in the composition of the blood produced by the low state of vitality, and in a great measure determined by the course of the disease. It is true that in many instances fibrinous

<sup>1</sup> Wilks and Moxon.

clots are found, in which deposition has taken place on the surface of a dark blood clot. But these are pathologically and clinically distinct from the class of cases under notice. And even in these, weakness of the circulation does not appear invariably to be the cause of the primary dark clot. Thus in a case which came under my notice some months ago, the patient had been able to lead a very active life till the day before his death. He was a travelling pedlar, and came to me complaining only of præcordial pain. He had walked nine miles that day. The pulse was 110, feeble; the heart sounds normal, but weak. He died suddenly next morning, when the left ventricle was found to be almost completely filled with a firm organised concretion, the centre of which contained softened remains of a large dark clot. The man had been in good health previously, and could not account for the onset of his malady.

It is urged in support of the stagnation of blood theory that the clots are larger and more frequently found in the right cavities than in the left, the reason adduced being that the circulation is feebler on the right side, on account of the thinner walls of these cavities. In a case, however, of congenital malformation which came under my care a few weeks ago, the ventricular walls were equal, while the right auricular wall was very much thicker than the left. It was one of stenosis of the pulmonary artery, with absence of the upper part of the septum ventriculorum. The aorta was very much enlarged, and arose from both ventricles. The foramen ovale was patent, but there was no open ductus arteriosus, on account of the aorta passing over the root of the right lung. Death took place very slowly in this case, and on examination firm fibrinous coagula were found in the right auricle and ventricle. The ventricular clot was prolonged into both the pulmonary artery and the aorta. The left cavities contained only fluid blood. This case is interesting, as it shows that weakness of the heart's action alone is not the cause of the deposition, or it would have occurred in both sides alike, and also that in this malformation admixture of the venous and arterial blood does not take place within the heart to any great extent.

The symptoms which these ventricular clots may produce will be described in the following cases:—

*Case I.*—Walter G., aged 30, had suffered from cardiac disease for eighteen months. The symptoms complained of were bad digestion, dyspnoea on exertion, and swelling of the ankles at night. The heart's action was moderate in strength, the area of dulness slightly enlarged. The apex beat was displaced downwards, the impulse increased, with a distinct thrill on palpation. A loud harsh systolic bruit was heard over the whole of the præcordia, loudest at the apex, and transmitted towards the axilla.

On the 22nd of March, the patient's friends sent for me suddenly, saying that he was dying. On my arrival I found him just recovering from a paroxysm of pain in the cardiac region, which had seized him as he rose from the sofa and attempted to go upstairs. This pain was accompanied with a feeling of impending death, which did not wear off until some time after the pain had ceased. There was great difficulty of breathing during the paroxysm. The friends stated that the attack lasted for about ten minutes, and then gradually ceased. At the time of my visit the heart was beating rapidly and somewhat feebly. The character of the sounds were not altered.

About three hours afterwards the patient had a similar attack, which proved fatal in a few minutes.

At the *post-mortem* examination, the heart was found moderately hypertrophied. The mitral valve showed old standing lesion, which had occasioned the insufficiency detected during life. All the cavities were moderately filled with fluid and clotted blood. The left ventricle contained, in addition, a firm fibrinous coagulum, adherent to the mitral valve and to the anterior and left sides of the cavity. It extended for several inches along the aorta, and presented the markings which I have described as being characteristic of these clots. There was no engorgement of the lungs.

*Case II.*—Peter S., aged 34, a pointsman, was admitted into the North Staffordshire Infirmary on 11th October 1877, suffering from laryngeal phthisis. The disease had existed six months. Patient pale and emaciated; almost complete aphonia; cough very slight; expectoration scanty. Evening temperature, 100°; pulse rather rapid. Patient able to walk about every day. The physical signs showed disease in both apices, in addition to the laryngeal affection.

On the 20th of October, nine days after admission, patient was suddenly seized with pain in the region of the heart and a feeling of suffocation while raising himself in bed. He grasped the sides of the bed, and leant forward with an expression of extreme anxiety and fear. The countenance was pale and covered with clammy sweat. The heart sounds were weak and muffled; the pulse rapid, feeble, and irregular. The attack lasted for nearly a quarter of an hour, when it gradually wore off, but the anxious expression remained. An hour and a half afterwards the patient experienced a similar attack, but not quite so severe.

On the 21st October, patient had a severe attack, which lasted about eight minutes.

On the 22nd October, while sitting up in bed talking to the nurse,



the patient suddenly pressed his hand over the region of the heart and expired.

Between these attacks the pulse almost regained its previous strength. No bruit could be detected, although the sounds were rather weak. The patient never lost the dread of death after the first attack.

After death the heart was found natural in size, with all the valves healthy. All the cavities contained dark soft blood-clot. A firm greyish-coloured fibrinous clot was found adhering to the wall of the left ventricle along nearly the whole length of the cavity, and to the chordæ tendinæ of the mitral valve. It was prolonged for three inches into the aorta. Several small cavities were found in the left lung. The right lung was loaded with grey miliary tubercles. The larynx showed extensive tubercular disease.

*Case III.*—John S., aged 42, a wire-worker, was admitted into the North Staffordshire Infirmary on the 7th May 1879. He was suffering from advanced phthisis of several years' duration. The cardiac dulness was increased downwards and inwards. A loud friction sound was heard over the whole area. The heart's action was rapid, the pulse firm.

May 13th.—Action still rapid, friction sound gone, systolic bruit at base.

May 24th.—Patient died suddenly while raising himself in bed.

*Post-mortem* appearances. — Heart hypertrophied; pericardial adhesion; valves competent, but thickened and enlarged. The right cavities were filled with large dark clots. The left auricle contained fluid blood. A fibrinous clot (fig. 3, Plate IX.) was found in the left ventricle, adherent to the left side of the cavity and to the mitral valve. It extended into the aorta, but this portion was unfortunately cut off about 2 inches from its origin in removing the heart from the body.

The chief symptom observed in cases I. and II. was the great anxiety and dread of death exhibited by the patients during, and for some time after, the attacks. They kept continually repeating, "Oh, I cannot live!" The pain was not so severe as that observed during the paroxysms of angina pectoris, to which these attacks bore considerable resemblance. In angina there are two pathologically distinct elements—neuralgic pain and disturbed muscular action. The former is the characteristic symptom; the latter may be entirely absent. In these cases, on the contrary, disturbed muscular action was the only, or at any rate the predominant, source of the pain and distress. I am doubtful whether there was any neuralgic element associated with the paroxysms. The pain had not the sharp radiating character of neuralgia, but resembled that of muscular spasm. Professor F.

Ogston of Aberdeen informs me that, in his opinion, the heart pang observed in many instances just before death is due to heart-clots.

There was no true dyspnœa observed in these cases. The breathlessness appeared to be voluntary, the patient seizing hold of the sides of the bed, and leaning forward to obtain relief. And there was no stoppage of the circulation, for the clots were not large enough to block up the aorta. Moreover, the surface of the body was pale, and not livid, as would have been the case had there been true dyspnœa.

Flint says :—"The disturbance of the heart's action explains certain symptoms which frequently enter into severe paroxysms of angina, namely, the sense of impending dissolution and the necessity of maintaining immobility of position. The danger depends on the disturbance of the heart's action." There can be no doubt but that this was the cause of death in the cases recorded above. In each the first attack began immediately after the patient assumed the erect posture. The heart, beating quietly as long as the patient was recumbent, had been thrown into more vigorous action by the change of position, and the ventricle, attempting to propel the blood more forcibly through the aorta, had been arrested in its contraction by the presence of the concretion in its interior. The clot had therefore acted as a foreign body, and induced the fatal spasm.

The third case differed from the preceding, as the concretion gave rise to no disturbance of the heart's action nor other symptoms during life, although it was undoubtedly the cause of the sudden death. It was the only case, too, where physical signs denoted the existence of the coagulum. The large aortic clot produced the basic systolic bruit heard for eleven days before death. This murmur might have been detected earlier had it not been for the loud pericardial friction sound.

[EXPLANATION OF PLATE.

## EXPLANATION OF PLATEIX.

Fig. 1, A. Flat fibrinous concretion in left ventricle, attached by base to the anterior flap of the mitral valve. Fig. 2, A. Flat concretion in left ventricle attached to mitral valve, and prolonged for 10 inches into aorta.

Fig. 1, B. Concretion attached to mitral valve, and extending towards apex. (This clot was firmly adherent to the aortic valves.)

Fig. 2, B. Concretion attached to mitral valve, adherent along anterior and left side of ventricle, and joined to the auricular clot (A C).

Fig. 1, C. Concretion from left ventricle, with part of auricular clot (A C) attached. Fig. 2. Right ventricular and portion of right auricular clots from same case. Fig. 3. Left ventricular concretion; heart much enlarged. Fig. 4 and fig. 4'. Left ventricular clot from case of mitral stenosis, with hypertrophy.

*Note.*—All these figures are two-thirds natural size.

**A SIMPLE METHOD OF DEMONSTRATING THE NERVES OF THE EPIGLOTTIS.** By WM. STIRLING, M.D., Sc.D., *Professor of the Institutes of Medicine, University of Aberdeen.*

(From the Physiological Laboratory of the University of Aberdeen.)

THE stratified epithelium covering the laryngeal surface of the Epiglottis is remarkably transparent, so that one can see deeper seated structures through it. With Mr. George Duffus, Student of Medicine of this University, I had occasion to investigate the distribution of the branches of the superior laryngeal nerve upon the laryngeal surface of the epiglottis. All that is required to show the course of the fibres of this nerve is to expose the laryngeal surface of the epiglottis to the vapour of a one per cent. solution of osmic acid for a short time. Within a few minutes a beautiful plexus of black lines stands out clearly and distinctly. Usually there is a moderately strong trunk running up pretty near the margin of the epiglottis, and from this many fine branches are given off, which unite with similar branches from the corresponding branch on the opposite side, and thus a very dense plexus of medullated nerve fibres is formed immediately under the epithelium.

This simple and easy method affords a satisfactory mode of demonstrating the singular richness of the nervous supply of the epiglottis. Of course this method reveals only the medullated fibres, but these are very numerous. Their actual mode of termination must be determined by other methods. The demonstration can be done so rapidly that it is one well suited for class purposes. The only precaution to be observed is not to expose the epiglottis too long to the action of the vapour of the osmic acid. The nerves to the epiglottis reach it near its base, and pass to its laryngeal surface; some of the branches terminate in connection with the taste bulbs which exist on that surface, and some in connection with the glands which lie immediately under the epithelium. The extreme density and closeness of the plexus explains the remarkable sensibility of the laryngeal surface of the epiglottis. The arrangements are easily made out in the rabbit, rat, cat, dog, sheep, or ox, and the epiglottis of a sheep which has been dead for a day or two does quite well for the purpose. A careful examination of the nerve before it reaches the epiglottis reveals the presence of numerous ganglionic cells.

## THE TRACHEALIS MUSCLE OF MAN AND ANIMALS.

By WILLIAM STIRLING, M.D., SC.D., *Professor of the Institutes of Medicine, University of Aberdeen.*

(From the Physiological Laboratory, University of Aberdeen).

THE trachealis muscle, which completes the trachea behind, varies somewhat in its arrangement in different animals. In man, the continuous layer of non-striped muscle, which forms this muscle, is described in Quain's Anatomy as passing "not only between the ends of the cartilages, but also opposite the intervals; those opposite the ends are attached to the ends of the latter, and encroach also for a short distance on their inner surface." "Outside the transverse fibres are a few fasciculi, having a longitudinal direction." A careful examination of a number of transverse sections of the trachea shows that the attachment of the muscle encroaches for some distance upon the inner surface of the rings, where it is firmly attached to the perichondrium. The longitudinally arranged fibres are seen in several bundles outside the transversely disposed fibres. In a section which has been stained with picrocarmine, the different arrangement of the fibres is easily made out, for all the connective tissue becomes of a bright red, while the non-striped muscle is reddish brown. The acini of many of the mucous glands lie outside the trachealis muscle, so that their ducts have to pierce it to open into the trachea.

In the *cat*, the trachealis muscle consists of fibres arranged transversely. They are attached to the *external* surfaces of the cartilages at a considerable distance from their free posterior extremities, so that they can thus exert considerable action. They also exist between the cartilages, and are attached to the upper and lower margins of adjoining cartilages. The fibres are very firmly adherent to the perichondrium. There are septa of connective tissue, which envelope bundles of the transversely directed fibres, so that on making a vertical section of a trachea posteriorly in the region where the trachealis muscle is attached to the cartilage, the muscle is seen cut transversely in small blocks, each surrounded by its own perimysium. In the *cat*

when the trachea is separate from its anatomical connections, a vertical section shows that the thin edges of the cartilages overlap, and the muscle has such wide attachments that the free ends of the rings also overlap, and the mucous membrane is thrown into longitudinal folds, projecting into the trachea. In such a section one almost invariably meets with a section of a small microscopic nerve ganglion. These ganglia are extremely numerous in the course of the branches of the recurrent laryngeal nerve, where they are distributed to the trachealis muscle. Indeed, there is a very distinct plexus, containing heaps of ganglionic cells at the nodes, not only in the cat, but in many other animals.<sup>1</sup> This plexus, in all probability, is comparable to the nerve-plexuses that occur in the walls of the intestine. The mucous glands of the trachea of the cat are very numerous, and, as is usually the case, they are more numerous between two cartilages than immediately between the body of the cartilage-hoop and the mucous membrane. The acini of these glands contain cells which are usually far more "granular" in their characters than the corresponding glands of the dog.

In the *dog*, the trachealis muscle presents much the same arrangement as in the cat, its attachment being also to the *external* surface of the cartilages, and also extending a considerable distance forwards on the cartilage. The muscle does not seem relatively to be so thick as in the cat. The mucous glands are quite different in their characters from those that exist in the cat. They usually have clearer epithelium, more like true mucous glands, and they are not so numerous as in the cat's trachea. The ends of the cartilages overlap when the trachea is removed from its connections. A very few longitudinally disposed fibres exist outside the transverse ones. Neither in the dog or cat do any gland acini occur outside the tracheal muscle, but there are numerous nerves and nerve ganglia.

In the *rabbit*, the muscle is also attached *externally*, and the same arrangement exists in the *rat*, and in both cases the fibres are directed transversely; while in both the attachment includes about one-fifth or thereby of the total circumference. The cartilages overlap at their free ends, so that there are three

<sup>1</sup> William Stirling, *Text-Book of Histology*, p. 59; Kandarazki, *Archiv. f. Anat. u. Physiologie*, 1881, p. i.

or four longitudinal folds of mucous membrane projecting into the trachea to be seen in a transverse section. In a trachea of a rat, slit open vertically in front and spread out, the arrangement of the fibres is easily seen. The fibres form a complete layer, and are attached to the upper and lower borders of the cartilages as well as to their posterior surface. The ganglionic nerve plexus is particularly abundant in the trachealis muscle of the rat.

In the *pig*, an entirely different arrangement prevails. The trachealis muscle is attached to the *internal* surfaces of the cartilages at a considerable distance—about one sixth of the circumference—forwards from the free tips of the cartilage. The result is that in a section of the trachea of the pig, when it is severed from its connections, the trachealis muscle, with the mucous membrane covering it, runs across the lumen of the trachea, and divides into an anterior larger part and a posterior smaller one, which latter is bounded laterally chiefly by the projecting free ends of the cartilages which meet behind. This is an exaggerated condition of what obtains in man. In the *sheep* also many of the acini of the mucous glands lie outside the trachealis muscle, so that it is perforated by their ducts. In E. Verson's article on the larynx and trachea,<sup>1</sup> the transverse muscular layer of the trachea is represented as being inserted in the case of the sheep into the "external surfaces" of the cartilages. This is certainly a misprint for internal. In the *ox* also the muscle is attached to the perichondrium covering the inner surfaces of the cartilages, and the cartilages with their slightly everted tips<sup>2</sup> meet behind.

<sup>1</sup> *Stricker's Histology* (New Sydenham Soc. Trans.), vol. iii. 46.

<sup>2</sup> *Todd's Cyclopædia*, article "Respiration."

THE SULPHOCYANIDES OF AMMONIUM AND POTASSIUM AS HISTOLOGICAL REAGENTS. By WILLIAM STIRLING, M.D., Sc.D., *Professor of the Institutes of Medicine, University of Aberdeen.*

(From the Physiological Laboratory of the University of Aberdeen.)

RECENTLY, in conjunction with Mr. Arthur Rannie, M.B. and C.M. of this University, I had occasion to investigate the action of a large number of substances upon the coloured blood corpuscles of the frog and newt. Amongst other substances, we investigated the effects of a 10 per cent. solution of the sulphocyanide of ammonium and the corresponding salt of potassium.

*Action on Blood Corpuscles.*—On adding a drop of either of the above solutions to a drop of blood of a newt or frog, but preferably the former, after a short time the coloured blood corpuscles begin to undergo a remarkable change. They give off from their margins fine granules of hæmoglobin, which can be seen detaching themselves and dancing about in the surrounding fluid. Often a thin narrow thread is seen to attach a granule. This thread is severed, and the granule is free. At other times the whole outline of the corpuscle changes in shape, and now and again larger masses of the peri-nuclear part of the corpuscle are severed from the parent mass. The hæmoglobin is often heaped at two masses at the ends of the corpuscle, or it may be at the sides. So far, these effects closely resemble those produced by urea and some other substances, such as dilute sherry wine (Wm. Addison) and the aqueous extract of putrid muscle (Dowdeswell). After a time, however, the hæmoglobin becomes quite discoloured or removed, and coincident with this, remarkable changes occur in the nucleus; it swells up, becomes more distinct, and there is revealed in its interior an exquisitely arranged intra-nuclear plexus of fibrils. Some interfibrillar material seems to be acted upon, and swells up, so as to separate the fibres that form the meshwork, and so to alter their refractive index, that their arrangement can easily be made out. This plexus can be stained with fuchsin or eosine. The plexus thus revealed can be kept for a long time. It is very resistant, and



defies putrefaction for a long time, even for several months; for I have often seen it in a mixture of blood and sulphocyanide solution, which was everywhere permeated with fungi, but still the plexus stood out boldly in the field, unaffected by the decomposition that had taken place in the surrounding mixture. I have not described all the effects of the sulphocyanides on blood, which I have done elsewhere along with Dr. Rannie, but I desire to set forth the value of the sulphocyanides as histological reagents, admirably adapted for revealing the presence and arrangement of the intra-nuclear plexus of fibrils in coloured and colourless blood corpuscles.

If a small quantity of blood be mixed in a glass capsule with the sulphocyanide solution, the whole assumes a semi-gelatinous consistence, and the physical characters of it closely resemble moderately thick mucus. Can this appearance have any relation to the presence of a mucin-like substance which L. Brunton<sup>1</sup> found to exist in the nuclei of the coloured blood corpuscles of birds? Experiment must determine. At any rate, the appearance is very characteristic.

*Action on Epithelium.*—I find that a 10 per cent. solution of sulphocyanide of ammonium or potassium is an admirable "dissociating" medium for isolating epithelial cells. Small pieces of the tissue are placed in the solution for twenty-four or forty-eight hours. They may be stained afterwards with picrocarmine, but before doing so it is necessary to remove all traces of the sulphocyanide by steeping the tissue in water for a short time. The sulphocyanide causes a precipitation of the picric acid.

I have tried the effect of this reagent upon the mucous membrane of the stomach, small intestine, bladder, eye, and skin, and on membranes covered with ciliated epithelium, of the newt and frog, and in all these cases one can isolate the constituent cells with ease. The cells all show very distinctly an intra-nuclear plexus of fibrils. What seems to happen is this, that the inter-fibrillar ground substance of the nucleus swells up slightly, and so opens out the network of fibrils.

In the liver of the newt and frog an intra-nuclear plexus of fibrils is also revealed by similar treatment. The liver of the

<sup>1</sup> *Journ. of Anat. and Phys.*, iii. 91.

newt, however, is to be preferred. In the liver cells of the newt, which, like the cells of other animals, are often fatty, one may frequently observe a distinct envelope surrounding each cell, more especially after the prolonged action of the reagent. Of course, it might be said that this envelope was due to the action of the reagent, but we have the distinct proof afforded by Dr. Haycraft, that the liver cells have a wall which can be ruptured by pressure. In the case of the liver cells of the newt, one can also readily observe the nuclear membrane, and after the action of picrocarmine, the nuclear membrane and the plexus become stained red. Not unfrequently the liver cells of the newt, and also of the rat (where the cells, however, are much smaller) contain two nuclei, as has been observed by Asp<sup>1</sup> in the liver cells of the rabbit.

*Action on non-striped muscle.*—I employed the bladder of the frog and the mesentery of the newt, as recommended by Klein. In both cases one obtains a good view of the intra-nuclear flexus in the fusiform non-striped muscle cells, and the effect is improved by staining with picrocarmine, when the peri-nuclear part becomes yellow, and the nucleus with its fibrils bright red.

*Action on striped muscle and nerve.*—Here also the intra-nuclear plexus in the nuclei of the muscles (more especially of the newt) and those of nerve fibres, are brought out. Not unfrequently in the muscles of the newt, which are admirably adapted for showing the plexus, one observes several nuclei arranged in line, in a chain as it were, in the long axis of the fibre. These nuclei are produced by the transverse cleavage of an original nucleus. The plexus is found to exhibit a different arrangement in different nuclei, i.e., the nuclei are in different phases of development corresponding to the different stages described by Flemming and others. The arrangement of the fibrils can be made out quite distinctly without the use of staining reagents, as its refractive index is quite different from that of the surrounding sarcous substance.

*Action on cartilage.*—If the thin cartilage of the sternum of the frog or newt be used, it is easy to note a similar structure in the nuclei of the cartilage cells.

*Action on the crystalline lens.*—The effect on the fibres of the

<sup>1</sup> Ludwig's *Arbeiten*, viii. (1873), p. 124.

lens is quite different from that of any other tissue. The lens fibres, after being acted on for twenty-four or forty-eight hours, assume a beaded or moniliform appearance. Here and there are little bulgings on the side of the fibre, at tolerably regular intervals in some fibres, while upon others oval or rounded globules adhere in great numbers. All the fibres are not affected. In the fibrils we find rounded or oval globules, not unlike myeline drops, only they do not possess a double contour. As we know from the researches of Thin and Ewart,<sup>1</sup> that the lens fibres of the toad, frog, and some other animals are partially enveloped with a layer of squames, at first sight it might appear that the bulgings on the side of the fibres was due to a swelling up of these squames, under the influence of the reagent. On looking carefully at them, however, they do not contain a nucleus, and on some fibres they are very numerous and very irregular in size. Besides, in certain cases they become detached. They seem to be due to the action of the reagent upon some chemical constituent of the lens fibres. In some cases, however, the oval swelling on the side of a fibre seems to be distinct from the lens fibre, as when the fibre is seen on edge, it may be traced on one side of the oval mass, which is sometimes granular, but I have never seen a nucleus within it. Some of the swellings, therefore, may perhaps be due to the swelling up of the cells on the lens fibres.

*Action on the retina.*—After the retina of the frog has been subjected to the action of the sulphocyanide for twenty-four hours, the outer segments of the rods reveal transverse striation most distinctly, and the same transverse striation is admirably seen in the outer segments of the very large cones of fishes, such as the cod or haddock. Indeed, in these animals this transverse segmentation is easily obtained by a variety of reagents.

<sup>1</sup> *Journ. of Anat. and Phys.*, x. 223.

A NEW THEORY AS TO THE FUNCTIONS OF THE  
SEMICIRCULAR CANALS. By P. M'BRIDE, M.D.,  
F.R.C.P. EDIN.

As is well known the view now generally accepted as to the functions of the posterior division of the labyrinth, is that which has been so ably advanced by Mach, Breuer,<sup>1</sup> Crum-Brown,<sup>2</sup> Cyon,<sup>3</sup> and others. Briefly stated it amounts to this, that the semicircular canals are peripheral organs of a sense which enables us to estimate our position in space. That this is one of their functions is more than probable, but in the following pages it is proposed to show that to ascribe to them this physiological position alone is insufficient and inconsistent. It will, I suppose, be admitted that nothing in life is superfluous, that every physiological arrangement is the best possible for the well-being of the organism, and finally that in the animal economy there is a reason for every anatomical detail.

Starting from these premises then, I again say that the explanation hitherto advanced as to the functions of the semicircular canals, though probably correct so far as it goes, is quite inadequate to account for all the facts connected with their physiology. Let us glance for a moment at the anatomy of the internal ear.

The vestibule contains two sacs, an anterior and a posterior. These membranous bags are only connected indirectly through the two branches of the aqueductus vestibuli. The anterior or sacculus hemisphericus is, through the small canalis reuniens, continuous with the central duct of the cochlea, while the posterior or utricle is continuous with the three membranous semicircular canals. Both are surrounded, so far as they are unattached to their bony case, by peri-lymph and filled with endo-lymph. The stapes moves in the fenestra ovalis, which in the recent state it closes. This foramen opens into the posterior part of the vestibule, and therefore opposite the utricle.

Now, I ask, why this arrangement, if it be not essential for

<sup>1</sup> *Medizinische Jahrbücher*, 1874.

<sup>2</sup> *Journ. of Anat. and Phys.*, viii. 327.

<sup>3</sup> *Thèse pour le doctorat en Médecine*.

the proper fulfilment of function, that vibrations of the stapes should affect the utricle and canals equally with the cochlea?

Through their position, the utricle and canals are liable to be influenced by various diseased conditions of the ear. Pressure on the tympanic membrane, exudation within the middle ear, and loud sounds are all apt to be followed by giddiness; and this vertigo can only be explained on the hypothesis of the abnormal stimulation affecting that portion of the auditory nerve which supplies the ampullæ.

If the canals were concerned in equilibration alone, then, surely on general principles it is fair to assume that they would be placed in a position as safe as consistent with the proper fulfilment of their function. Now the function of equilibration could, it seems to me, be carried on just as well if they were altogether shut off from the auditory apparatus. We must, therefore, assume that they have another part to play in the animal economy.

It seems probable that this other function is to produce, through the ampullar nerves, reflex rotation of the head and eyes towards the point from which a sound proceeds, and that, further, the afferent impulse may, in the lower animals, co-ordinate and brace the muscles necessary for escaping from a danger of which sound is the first indication.

Although this view has never, so far as I know, been advanced by others, yet it derives support from the following passage in a paper by Högyes, on the "Causes of Vertigo from increased Intra-tympanic Pressure" (*Pflügers Archiv.* xxvi.). "These experiments of rotation and stimulation, he says, also prove that similar bilateral reflex connections exist between this peripheral end organ (semicircular canals) and other muscles of the body through certain centres of the cerebrum and medulla. This applies more particularly to the muscles of the head and neck, as also to those of the trunk and extremities."

It is a familiar fact that wild animals go through certain muscular movements on hearing any unusual sounds. Thus a whistle or shout will often bring a deer to a stand still, with ears pointed, head turned towards the source of sound, and eyes looking in the same direction. It is associated movements like these, which I believe to be reflex, and consider to be due to an

afferent impulse communicated to the nerve-endings in the semicircular canals.

Such movements are necessary for the safety of wild animals, for thus the ear is placed in a better position than before for appreciating a repetition of the alarm, the eyes are turned towards the source of danger, and the muscles of the trunk and extremities are braced and ready to aid in escaping. It is quite obvious how essential it is that such combined movements should be reflex. Were it not so, much valuable time would be lost before the impression could be analysed and converted into "out-going currents" to all the muscles required.

It is now desirable to enter somewhat more fully into arguments which support our theory.

I. First in order comes the fact that the semicircular canals are so placed that every sonorous vibration capable of being perceived as sound, must necessarily cause movement of the endo-lymph which they contain, and of the peri-lymph which surrounds them.

Let us just glance for a moment at the part played by the internal ear in the act of hearing. The stapes is pressed inwards and impinges upon the peri-lymph, the latter again reacting upon the endo-lymph. Now, according to Hensen (*Handbuch der Phys.*, Hermann.), "the labyrinthine fluid moves only as an absolutely incompressible mass, and is therefore incapable of vibrations; it is accordingly pushed onwards by the stapes as soon as a point of escape is presented." Hensen goes on to say that one such means of escape is to be found in the openings of the aqueductus vestibuli, and that in all probability, in this way, vortex movements are produced in the semicircular canals.

Now, it is well known that those observers who look upon the latter as organs of the "static sense" only, base their arguments upon the fact that movement of the endo-lymph produces afferent impulses through the nerves of the ampullæ. We have seen, however, that movement of the endo-lymph must occur at every act of hearing, and therefore, in like manner, afferent impulses will result.

That changes in the fluid contents of the internal ear are likely to stimulate the ampullar as well as the cochlear terminations of the auditory nerve, is rendered extremely probable by the similarity of nerve-endings in the two parts. In both the latter

are in the shape of slender hairs proceeding from modified cells, seeming certainly to indicate that their function is to react to similar stimuli. Those in the cochlea react to sound only, those in the canals react to sound but have also probably a secondary function connected with equilibration.

That loud noises affect the posterior as well as the anterior part of the labyrinth is rendered all but certain by the common occurrence of such cases as the one described by Brunner (*Zeitschrift für Ohrenheilkunde*, ix. 142), the patient getting giddy immediately after a rifle had been fired close to her ear.

II. My second argument is based upon the unnecessarily exposed position of the semicircular canals, were their function purely that of equilibration. To this point I have already shortly alluded, and have pointed out that this function could be just as well performed if a complete separation from the auditory apparatus existed. In all vertebrates, however, the utricle and saccule, where they exist, stand in much the same relation to one another as is the case in man and the higher animals—and even among the fishes a rudimentary cochlea exists.

There seems, then, to be a necessity for an intimate connection between these so-called organs of equilibration and the organ of hearing. As it is the pressure of a foreign body on the drum-membrane, obstruction of the Eustachian tube, inflammation of the middle ear, and even loud sounds are peculiarly apt to cause giddiness, which undoubtedly arise through changes in the endo-lymph. If the semicircular canals are situated in a position so exposed for no reason whatever, they present a striking contrast to all other physiological arrangements. On the other hand, if they be so placed—as I believe—for some useful purpose, it remains to be considered what that purpose is.

To my mind, the most probable explanation is, that while sonorous vibrations are conveyed by the cochlear nerve to the true auditory centre, an afferent impulse is at the same time sent to various motor centres through the ampullar nerves. How such an arrangement would assist wild animals in self preservation I have already suggested. It may be urged that the movements exhibited by deer and other animals after a shout or whistle are not reflex in the true sense of the word, but rather the result of an auditory impression. This view is also negatived by

the phenomena observed in a pigeon where the cerebrum has been removed. In such a case a pistol shot fired behind the animal will cause rotation of the head. That muscular movement often occurs synchronously with, or indeed rather before, the appreciation of the sound, must be well known to those who have ever experienced the starting so often produced by a loud noise. This familiar phenomenon—purely reflex as it is—certainly favours our theory, that while sonorous expressions are conveyed along the cochlear nerve to be perceived as sound, they are at the same time carried along the nerves of the canals and motor centre to be converted into muscular movements, and, so far as I have been able to observe, these latter occur only or principally on the side nearest the sound.

III. In the third place, the results obtained by experiment directly support the view that has been taken in this paper. In an elaborate article, Spamer<sup>1</sup> has proved from the results of experiments on pigeons that various stimuli—mechanical, chemical, and thermal—applied to the semicircular canals produce rotation towards the side stimulated. The only exception was in the case of electric stimulation, and the anomalous result in this case was probably due to deficient limitation of the current.

This author (Spamer) comes to the conclusion that movements towards the side operated on are the result of stimulation and not paralysis of the ampullar nerve. On this point he says: "The following experiment furnishes us with complete proof of the fact that the phenomena under consideration are the result of stimulation. Let one or both of the superficial canals on one side be cut through or removed, then touch the same canal or canals of the opposite side to the same extent with a red-hot metal sharply, or until they are actually burnt. As a result the tendency before existing to turn towards the side first operated on will have disappeared, and there exists a tendency to turn to the other side. At other times, the latter immediately follows the former, which only lasts for a few seconds."

This, then, in itself shows that stimulation of the canals will induce rotation towards the side stimulated. We have also proved that stimulation of the ampullæ is in all probability produced by sound waves. Therefore, it seems fair to conclude that

<sup>1</sup> *Pflüger's Archiv*. xxi.



there will be a tendency to rotate towards the origin of a sound, which must of course affect principally the auditory nerve on the side which is nearer the vibrating body. Again, patients who suffer from auditory vertigo have, so far as I have been able to observe, a tendency to stagger towards the side corresponding to the ear which causes the disturbance—in other words, towards that side on which the ampullæ are stimulated. I am aware that cases have been recorded in which this state of matters was reversed, so that to others this argument may not seem reliable. I can only say that, having paid particular attention to auditory vertigo, I have never seen a case in which there was an inclination to move towards the unaffected side. But, granting this sometimes to be the case, the explanation is easy. A subjective sensation of falling to one side will naturally call into action the muscular mechanism necessary to prevent it, which may sometimes produce movement or even falling to the opposite side.

We know from the numerous investigations of Cyon (*Thèse, op. cit.*), that section of each semicircular canal produces a distinct and characteristic form of nystagmus.

Högyes, however (*op. cit.*), has gone further, and asserts that the nerves of the semicircular canals are directly connected with associated movements of the eyeballs. He also describes a most interesting experiment. In a living rabbit, the mastoid fossa was opened. He then passed a thin glass tube into the perilymphatic space and sucked out the fluid. He then gently blew into the tube, so as not to force back all the perilymph, and immediately noticed bilateral movements of the eyeballs. A little more pressure produced marked nystagmus. "One can so regulate the pressure," he goes on to say, "that without nystagmus, simple movement is produced."

As to the direction of this movement he also experimented, but in a somewhat different way. He laid bare portions of the labyrinth, and, on touching the horizontal canal, found that both eyes were moved towards the operated side.

There is one more conclusion arrived at by Spamer (*op. cit.*), which I cannot omit. To quote his words, "the results (*of experiment*) compel us, as I think, to believe that in consequence of unilateral injury to one or several canals, the perception of the position of parts on the corresponding side (only or at least

chiefly) is disturbed, and moreover, at first (as a result of a stimulus starting from this point), more powerful impulses are sent to the muscles of the same side in flying and walking. Thus, the fact of the turning and circus movements in this direction can be accounted for."

Let us now consider our arguments, and the deductions to be derived from them. It has, I think, been shown (1) that the nerve-endings in the semicircular canals are stimulated more or less every time a sound is perceived; (2) that the effects of stimulation are rotation of the head, eyes, and body towards the stimulated side, with increased activity of the muscles of the side.

The form of stimulation which has been used by experimenters is for obvious reasons immensely more severe than that which would be caused by sound. Yet the results obtained render it extremely probable that the effect of even a slight stimulus would be to produce rotation of the head and eyes towards the side acted upon. Where sound is the stimulus, we should expect this movement of the head and eyes, with more or less increased muscular activity in the trunk and limbs on the corresponding side, denoted by a partial rotation of the body.

The position so produced corresponds almost exactly with that assumed by a deer startled by a whistle.

I cannot but think that the facts we have just considered, make out a strong case in favour of the theory adopted in these pages, viz., that one function of the ampullar nerves is to convey directly to motor centres stimuli, by the reflex action of which on the muscular system the animal is placed in a better position, (1) for appreciating a repetition of the sound, (2) for seeing the place in which a sound originates, (3) for escaping from the cause of alarm.

SOME POINTS IN THE MYOLOGY OF THE COMMON  
PIGEON. By WILLIAM A. HASWELL, M.A., B.Sc. (Edin.),  
*Demonstrator of Comparative Anatomy and Physiology in  
the University of Sydney.*

THAT there should be any moot points in the anatomy of such a common bird as the pigeon will, I think, hardly be credited by most. But in this, as in many other instances, we find that the object which lies within easiest reach has been to some extent overlooked. A careful examination some years ago of the muscular system of such species of pigeons as I could then procure, showed me that certain points in the myology of that bird had been overlooked by the general writers on avian anatomy, and though it seemed unlikely that my observations could be entirely new, it seemed to me that the importance in taxonomy of the points referred to had been overlooked. I, therefore, published in the *Proceedings of the Linnean Society of New South Wales* a short abstract of my observations (Notes on the Anatomy of Birds—III. The Myological Character of the Columbidae, vol. iv. pp. 303–310) in which I set down the following points in the myology as being characteristic of the pigeons:—

1. The absence of the posterior belly of the latissimus dorsi.
2. The expanded form of the tensor accessorius.
3. The absence of the gluteus externus and the presence of the adductores brevis et longus, the semitendinosus, and accessory semitendinosus.<sup>1</sup>
4. The special relation of the tendon of the ambiens (when present) to the fibular head of the flexor perforatus secundus tertii digiti.
5. The presence of muscles which seem to represent the lumbricales in the foot.

My attention has been directed anew to this subject by a note in a paper on *Pterocles*, by Dr. Hans Gadow, in Part II. of the

<sup>1</sup> The *seminembranosus* is inadvertently mentioned instead of the *accessory semitendinosus* in the short summary at the end of the paper. The former muscle is very constant in birds, being, however, absent, according to Sundewall, in some grebes.

*Proceedings of the Zoological Society* for 1882, wherein the author states that of these five points one is "totally incorrect," while three others are "not characteristic of the pigeons."

It will be well at once to point out that Dr. Gadow is mistaken in supposing that, when I speak of certain modifications as being characteristic of the pigeons, I intended it to be understood that they are *peculiar to* that family. It may seem strange that it should be necessary to point out this distinction to any one used to scientific phraseology, but it will be seen that Dr. Gadow has been misled by a neglect of it. But, first, as to the point which is "totally incorrect"—

The latissimus dorsi muscle in all birds, with the exception of a few pigeons, consists of two ribbon-like muscular bellies united by an intermediate fascia, wide apart at their origin, and converging towards the axilla; the front portion arises from the spines of three or four of the anterior dorsal vertebræ; and the hinder belly takes origin from the fifth and sixth dorsal spines and from the front margin of the ilium. It is very much, in fact, as if the middle portion of the latissimus of mammals had been cut out, leaving only the anterior and posterior fibres. This arrangement occurs, so far as my observations extend, in *all* birds, with the exception only of certain pigeons, including *Columba livia*, and *C. ænas* of Europe and certain nearly related Australian genera. In these the hinder belly is always entirely absent. At the time when I wrote the paper referred to on the myology of the pigeons, I had not had the opportunity of dissecting any fruit-pigeons, and I was led to suppose that this remarkable peculiarity extended to the whole family; but, more recently I have pointed out, in a note on the anatomy of *Edircinus* and *Turaccena* (*Proc. Lin. Soc. New South Wales*, vol. vii. p. 115), that in the fruit-pigeons the arrangement of the muscle is normal. Thus, though this exceptional arrangement is not found in all the Columbidae, its occurrence in this family alone of all birds is noteworthy, and it is a point which will probably prove of considerable taxonomic value within the limits of the family itself.

*The absence of the gluteus externus and the presence of the adductores brevis et longus, the semitendinosus, and accessory semitendinosus.*—With reference to the gluteus externus Dr. Gadow

states:—"Now the m. gluteus externus (= *gluteus anterior*) is generally very small, but plainly visible in many birds, such as pigeons, passerine birds, &c., and not absent as stated by Mr. Haswell." That this muscle, though well-developed in many families, is extremely small in others is a well-known fact; but in the pigeon it is *entirely absent* as a separate muscle, as very little trouble would have enabled Dr. Gadow to ascertain, and as has been remarked long ago by Garrod.<sup>1</sup>

As regards the other muscles, whose presence is noted, their importance in classification was pointed out by Garrod in a well-known paper ("On Certain Muscles of the Thigh of Birds and their value in Classification," *Proc. Zool. Soc.*, 1873, pp. 626-644, and 1874, pp. 111-123). The absence or presence of these muscles he regarded as being of some importance in determining the affinities of the various orders and families. The pigeons possess the whole four muscles, and this I put down as one of the noteworthy points on the myology of the family, adding—"The significance of these muscles has been pointed out by Mr. A. H. Garrod ('On Certain Muscles of the Thigh of Birds and their value in Classification,' *Proc. Zool. Soc.* 1873 and 1874)." It is, therefore, somewhat difficult to understand the meaning of the remark which Dr. Gadow makes on this point—"The four other muscles are well-developed in most birds, as Professor Garrod has stated over and over again, and as the dissection of any fowl will show."

*The special relation of the tendon of the ambiens when present, to the fibular head of the flexor perforatus secundus tertii digiti.*—On this Dr. Gadow remarks: "The distal end of the ambiens muscle, when typically developed, always forms the continuation of one of the heads of the m. perforatus dig. II. et III." This is, I believe, perfectly correct. But it does not contradict my statement. As a rule the fibres of the distal tendon of the ambiens become broken up when they reach the upper portion of the leg, and become scattered through the fleshy substance of the muscles referred to. In the pigeons the arrangement is peculiar in this, that the distal tendon maintains its consistency and joins the proximal tendon of the fibular head of the flexor perforatus secundus tertii digiti, so that the whole might be regarded

<sup>1</sup> See his Collected Scientific Papers, p. 240, or *Proc. Zool. Soc.* 1874.

as an ilio-phalangeal muscle with two bellies, and the intermediate tendon united to the fibula by a tendinous band.

*The presence of lumbricales in the foot.*—The muscle which I ventured, on account of its proximal attachment to the flexor tendons opposite the distal portion of the tarso-metatarsus, to regard as the equivalent of the lumbricales muscles of mammals, is a short and broad fasciculus of fibres which becomes bifurcated distally, and has its insertion into the sheaths in which run the flexor tendons for the second and third toes. Dr. Gadow states that this muscle was described, but not named, by Meckel in his *Vergleich. Anat.* Band III. p. 388, and his "*Archiv für Anat. u. Physiol.*," pp. 278 and 279, and that it is not peculiar to the pigeons, as it occurs in many other birds, "*e.g.*, the Ratitæ." I am, unfortunately, unable at present, by reference to the works cited, to check Dr. Gadow's identification of the muscle which I have called "*lumbricales*," with one described by Meckel; but the statement that the former occurs in "*many other birds, e.g.*, the Ratitæ," is certainly erroneous. It is not mentioned by Owen in his "*Memoir on the Apteryx*," in his article "*Aves*" in Todd's *Cyclopædia*, or in his "*Comparative Anatomy and Physiology of Vertebrates*," nor by Selenka in the "*Vögel*" of Brown's "*Thierreich*," nor by Alix in his "*Appareil locomoteur des Oiseaux*," nor is any mention made of it by Garrod in his papers on the "*Ostrich*," in which a full description is given of all the flexors of the toes. I have looked for it in many families of birds, and have only found it in one, viz., the fowls, so that its occurrence in the pigeons, though not strictly *peculiar* is yet to be regarded as highly *characteristic*.

THE ACTION OF SALINE CATHARTICS. By MATTHEW HAY, M.D., *Demonstrator of Practical Materia Medica in the University of Edinburgh* (PLATE X.).

(Continued from page 78.)

SERIES OF EXPERIMENTS, G.

The effect of the salt on the blood and the circulation ; as also its effect on the temperature of the body.

I HAVE already in Series C. drawn attention to the fact that the blood becomes concentrated for a short time after the administration of a strong solution of sulphate of soda. A second concentration, but of less degree, is observed several hours later. At neither stage, however concentrated the blood may be, have I ever perceived any crenation of the corpuscles, if they were examined immediately after the blood was taken from the body ; but, if the blood was allowed to remain for a few minutes beneath the cover-glass on the microscopical slide, I have often observed that the corpuscles of the concentrated blood became more or less crenated, whilst within the same time the corpuscles of ordinary blood did not exhibit the slightest appearance of crenation. This is a point almost devoid of interest, since the crenation does not occur within the body.

The supposed alteration of the proportion of the white to the red corpuscles produced by the administration of a saline purgative is of more importance, although the mode of its production and its effect on the body are quite unknown. I have myself made no observations relative to this alteration, as the distribution of the leucocytes in a drop of blood is so unequal that the average of a very large number of enumerations of the corpuscles is necessary to obtain even an approximate knowledge of their proportion in the blood. Ch. Robin, from direct observation, is of opinion that " a simple diarrhoea, such as that caused by the administration of a purgative, as Seidlitz water, suffices to produce a notable augmentation of the leucocytes."<sup>1</sup> On the other hand, Brouardel,<sup>2</sup> as the result of a number of experiments on

<sup>1</sup> Ch. Robin, *Op. cit.*—*supra*, vol. xvi. p. 440.

<sup>2</sup> Brouardel, *Op. cit.*—*supra*, vol. xvi. p. 440.

man, finds that after the administration of saline and other purgatives the absolute number of the leucocytes in the blood is sometimes diminished, at other times increased, but that in relation to the red corpuscles the number of the leucocytes is almost without exception diminished. The enumeration of the corpuscles in the blood from each experiment was made in ten different portions of the microscopic field by every member of Brouardel's histological class, each with a separate portion of blood, so that Brouardel's observations are as trustworthy as can be obtained by the method at our disposal. It is to be remarked that both Brouardel and Robin examined the blood several hours after the administration of the purgative, and in most cases during that period in which I observed the secondary concentration of the blood. It is, however, highly probable that the alteration of the numerical relation of the white to the red corpuscles is in no way connected with the condition of concentration, but proceeds from the presence of the purgative in the blood affecting the transformation of the white into the red corpuscle, or disturbing the production of the white corpuscle in those localities of the body where it is believed to originate; and it is equally probable that the change is gradually accomplished, and, therefore, will have produced no perceptible effect, during the stage of primary concentration.

The blood, so far as I have observed, suffers no other physical or chemical change, except the alteration in its composition due to the presence of the salt. It will be remembered that I suggested, as the result of the experiments of Series D., that during the first hour after the administration of the salt the blood is probably highly charged with the salt, and that later the salt in great part disappears, mainly on account of its reappearing within the intestines. I may here state that since these experiments were made I have chemically estimated the amount of the salt recoverable from the blood at various periods after its administration, but have not been able, so far, to obtain evidence of a marked variation in the quantity of the salt in the blood. The result of these and other experiments will form the subject of a future communication.

The change in the metabolism of the body, which is intimately associated with the action of the salt on the blood, will be considered along with the effect of the salt on the urinary secretion.



There is still another action, or supposed action, of the purgative, which may be appropriately considered in connection with the blood. Does the administration of a saline cathartic affect the temperature of the body? Purgatives, and particularly saline purgatives, have long been used in the treatment of fever, under the impression that, among other effects, they help to cool the body. For the purpose of ascertaining if a saline cathartic actually reduces the temperature of the body, I made two experiments on man, a third on a cat, and a fourth on a rabbit. Although not sufficient in number to furnish a perfectly definite conclusion, yet they present fairly substantial grounds for inferring that the salt has little effect on the temperature. In two of the experiments, there was administered on the previous day, with the object of learning the mere effect of the cold water of the salt solution, a quantity of cold water equal in bulk and temperature to the salt solution given. The two individuals submitted to the experiment partook of breakfast at 8.30 A.M., and received no food until 4.30 P.M. In the case of the cat and the rabbit, they had no food on the day of the experiment until after eight in the evening. And on those days on which the

*Experiment CVIII.**Experiment CIX.*

A.B., set. 26. Temperature of Room, 10°-11° C.						C.D., set. 46. Temperature of Room, 16° C.					
First Day.		Second Day.		Third Day.		First Day.		Second Day.			
Normal.		Effect of Water.		Effect of Salt Solution.		Normal.		Effect of Salt Solution.			
Time.	Temp.	Time.	Temp.	Time.	Temperature.	Time.	Temp.	Time.	Temperature.		
A.M.	°C.	A.M.	°C.	A.M.	°C.	A.M.	°C.	A.M.	°C.		
10.30	36.2	10.40	35.9	10.20	36.0	11.45	36.9	11.25	36.7		
P.M.		P.M.		P.M.		P.M.		P.M.			
12.5	36.3	12.20	36.1	12.5	36.2	...	...	12.10	36.7	Salt solution.	
2.0	36.3	2.10	36.3	2.0	36.1	...	...	12.15			
...	...	2.28	36.3	...	...	...	...	12.30	36.6		
2.30	36.2	2.30	Water.	2.30	Salt solution.	...	...	12.45	36.65		
...	...	2.35	34.6	...	...	1.0	36.8	1.0	36.65		
3.0	36.3	2.40	35.7	2.40	35.5	...	...	1.30	36.7		
...	...	3.30	36.2	3.40	35.8	2.0	36.7	2.30	36.7		
4.30	36.1	4.30	36.2	4.25	36.1	3.0	36.6	3.30	36.7		
9.0	36.3	9.0	36.4	9.0	36.2	4.0	36.6	4.30	36.6		
...	..	...	...	...	...	8.30	36.9	8.30	36.5		

course of the normal temperature was obtained, and the course of it as affected by a drink of cold water, the same restrictions as to diet were imposed. The temperature was taken beneath the tongue of the men, and in the rectum of the rabbit and cat, and two observations were in most cases made at each period, the one to correct and confirm the other.

In Experiment CVIII, 600 c.c. (20 oz.) of water at 5° C. were given on the second day at the time indicated; and 15 grammes (fully  $\frac{1}{2}$  oz.) of sulphate of soda, dissolved in 600 c.c. of water, the solution having a temperature of 5° C., were administered on the third day. In the other experiment, 28 grammes (1 oz.) of sulphate of magnesia, dissolved in 250 c.c. (fully 8 oz.) of water, the temperature of the solution being 2° C., were given on the second day.

*Experiment CX.**Experiment CXI.*

RABBIT, male; 1.7 kilograms. Temperature of room, 10° C.						CAT, male; 3.1 kilograms. Temperature of room, 10° C.					
First Day.		Second Day.		Third Day.		First Day.		Second Day.			
Normal.		Effect of Water.		Effect of Salt Solution.		Normal.		Effect of Salt Solution.			
Time.	Temp.	Time.	Temp.	Time.	Temperature.	Time.	Temp.	Time.	Temperature.		
A.M.	°C.	A.M.	°C.	A.M.	°C.	A.M.	°C.	A.M.	°C.		
11.0	38.3	11.10	38.4	11.10	38.1	11.15	38.8	11.20	38.5		
P.M.		P.M.		P.M.		P.M.		P.M.			
...	...	1.40	38.4	1.25	38.2	12.50	38.6	1.35	38.4		
...	...	1.50	Water.	1.45	Salt solution.	2.20	38.7	2.20	Salt solution.		
2.0	38.2	2.10	38.2	2.15	37.7	...	...	2.45	38.1		
...	...	...	...	2.25	37.8	...	...	3.15	38.2		
...	...	2.45	38.4	2.50	37.9	3.40	38.5	3.45	38.6		
...	...	3.30	38.5	3.50	38.2	...	...	4.15	39.0		
4.0	38.1	...	...	4.10	38.6	5.30	38.6	5.15	39.3		
...	...	...	...	5.10	38.3	...	...	6.25	39.4		
6.0	38.3	...	...	6.20	38.5	...	...	7.35	39.2		
8.0	38.1	7.0	38.8	7.25	38.5	...	...	...	...		

In both experiments the salt administered was the sulphate of soda, and in equal strength of solution, namely, 10 per cent., and of like temperature—7.5° C., 50 c.c. having been given to the rabbit, and 40 c.c. to the cat. 50 c.c. of water, of the same temperature as the salt solution, were administered at 1.50 P.M. on the second day of Experiment CX.

In all these experiments a purgative discharge did not occur

during the period in which the temperature was recorded, but in all of them, except that with the rabbit, free purgation followed in the course of the evening.

Before drawing any conclusion from these experiments, I have to remark that, from several unrecorded observations of the daily course of the normal temperature of the animals employed, I found that the temperature of the same animal often varied very considerably from day to day, as also throughout any single day. Too much importance is not, therefore, to be attached to the variations observed during each experiment. We may, however, reasonably conclude from these experiments that the solution of the saline cathartic generally produces, almost immediately after its administration, a slight reduction of temperature, which never amounts to more than half a degree Centigrade, or about one degree Fahrenheit, and which continues, gradually becoming less evident, from one to two hours. The temperature having reached the normal, remains at this, or, if anything, mounts higher (Exps. CX. and CXI.). It is probable, however, that the later rise of temperature observed in these experiments was due to causes other than the administration of the salt. In certain circumstances, as in Exper. CIX., the temperature is scarcely affected; this is probably exceptional. Water, it will be observed, given in a quantity, and of a temperature, equal to those of the salt solution, also distinctly lowers the temperature of the body, but barely to the same extent as the salt solution, and evidently not for so great a length of time.

A saline cathartic would, therefore, appear to exert no, or very little, lowering effect on the temperature beyond that produced by an equal quantity of cold water. The slightly greater effect of the saline solution and its more protracted action are probably to be attributed to the absorption of heat which occurs when the solution is diluted by its mixture with the fluid contents or secretion of the alimentary canal and with the blood, a solution of a salt absorbing heat, or being reduced in temperature, when it is diluted.

But, although the degree of the general temperature of the body, or the temperature of the internal organs, is not affected by the salt, it seems almost to be otherwise with the absolute

heat or caloric of the body. For it is common experience that for several hours after the administration of a saline cathartic, and for many hours beyond the actual observed depression of temperature, there is a decided feeling of chilliness, sometimes accompanied by slight shiverings, the extremities often becoming extremely cold. This is not a mere subjective sensation of cold; for it is not difficult to ascertain that the temperature, for example, of the hands, is much lower than usual. Yet the temperature of internal and unexposed parts remains high. This loss in the absolute heat of the body is probably caused by a contraction of the peripheral arteries due to the slightly irritating action of the salt dissolved in and circulating with the blood. A full supply, therefore, of warm blood is denied to the peripheral parts of the body, and these being exposed to the cold of the surrounding atmosphere quickly lose their heat.

In so far, therefore, as its effect on the temperature is concerned, the advantage of the employment of a saline cathartic in the treatment of fevers is extremely doubtful; for, although the peripheral temperature is reduced, the temperature of the blood and of the viscera, on which the effect of a high temperature is most dreaded, is not lowered.

The supposition made in explanation of the salt reducing the temperature of the periphery of the body, namely, that the arterioles have their calibre diminished, receives support from observations I have made of the action of the salt on the circulation, to the consideration of which I now proceed.

In estimating the effect of the saline cathartic on the circulation I have confined myself to observing the effect on the pulse by means of Marey's sphygmograph. No direct experiments were made as to the action of the salt on the actual arterial blood-pressure, owing to the fact that, from sphygmographic observations, I knew the effect on the circulation was very gradually produced, and was probably at no time very marked; and in such a case I believed that the effect of the prolonged subjection of the animal to the requirements of a blood-pressure experiment, and the disturbance produced by the necessary administration of anæsthetics, would almost certainly obscure the action of the salt.

The prevalent belief has long been that cathartics, salines in-

cluded, lower the blood-pressure. And it is more with this object than from any anticipated effect on the temperature that cathartics have been employed in the treatment of acute febrile and inflammatory conditions, it being supposed that, by removing through the intestines from the blood a quantity of its fluid, they accomplish a form of depletion which results in a diminution of the volume of the blood and consequently of its pressure. This supposition is based on the erroneous assumption that a very gradual and not too extensive depletion of the blood is followed by a lowering of the blood-pressure. Still it is possible that, quite apart from this supposition as to the method of its production, cathartics lower the blood-tension. Lauder Brunton<sup>1</sup> gives two sphygmographic tracings of the pulse of a healthy man, one taken before, and the other after, the action of a purgative, which lend support to the common belief. The name of the purgative used is not stated. My own observations are more numerous than those of Brunton, and in their results they seem to contradict those of that eminent pharmacologist. I, of course, employed saline purgatives, and observed their effect on the tension and rate of the pulse, and, as in Lauder Brunton's experiment, in individuals with a normal circulation. The observations were made on the patient submitted to Experiments LIV. and LV., in both of which sulphate of soda was the salt employed, and on a second patient in the wards of the infirmary to whom a dose of sulphate of magnesia was administered. The sphygmographic tracings from the former patient were taken whilst the previous experiments were in operation.

*Experiment CXII.*—J. W., æt. 33, suffering from a chronic nervous disease of an ataxic character; general health otherwise good. The patient was placed in bed about twenty minutes before the first tracing was taken, and was kept in bed throughout the experiment. The sphygmograph (Marey's) was fixed with a comparatively light pressure over the left radial artery at the wrist, the arm being kept all the while steadily in the same place on the bed, and the sphygmograph was not moved from its original position until all the tracings had been taken, except that, immediately after taking the last tracing of the series presented, it was moved gently about for the purpose of ascertaining if the character of the tracing was not due to a change having occurred in the position of the instrument. But, although

<sup>1</sup> Lauder Brunton, *Practitioner*, vol. xii., 1874, p. 417.

several other tracings were taken, they did not differ in character from the one first procured. No food was given during  $4\frac{1}{2}$  hours previous to the administration of the purge—21·3 grammes ( $\frac{3}{4}$  oz.) of sulphate of soda dissolved in 85 c.c. (3 oz.) of water, or nearly a 20 per cent. solution of the salt. Purgation occurred next morning, fully 14 hours afterwards. A large series of tracings were taken, a few of which are reproduced. The following letters refer to those of the accompanying lithograph (Plate X.).

A. 10 minutes <i>before</i> the administration of the purgative.	Pulse, 60 ; resp. 20.
B. 60 minutes <i>after</i> "     "     "     "	Pulse, 56 ; resp. 20.
C. 125 minutes     "     "     "     "	Pulse, 60 ; resp. 26.
D. 185 minutes     "     "     "     "	Pulse, 56 ; resp. 24.

A tracing was taken  $9\frac{1}{2}$  hours after the administration of the purgative, the pulse being 52, and respiration 24 ; it was almost exactly the same in character as D.

*Experiment CXIII.*—Same individual as in previous experiment and conditions exactly alike, excepting that the salt was given dissolved in 454 c.c. (16 oz.) of water—about a 5 per cent. solution. A free watery purgation occurred two hours afterwards.

The series of tracings obtained during this experiment was similar in character to that of the previous experiment, except that the gradual change observed to occur in the form of the pulse-wave, although quite evident, was not so well marked. The rate of the pulse and the respirations was very little affected, falling very slightly after the administration of the salt, the former from 60 to 58, and the latter from 12 to 20.

*Experiment CXIV.*—W. F., æt. 19, convalescent from nephritis. Conditions were quite the same as in the previous experiments. The purgative administered was 21·3 grammes ( $\frac{3}{4}$  oz.) of sulphate of magnesia, dissolved in 227 c.c. (8 oz.) of water—about a 10 per cent. solution. Purgation took place  $2\frac{1}{2}$  hours afterwards, and was followed by another watery dejection a few hours later in the evening. The following numerals refer to those of the accompanying lithograph, in which a few of the sphygmographic tracings taken are represented.

I. 7 minutes <i>before</i> the administration of the purgative.	Pulse, 92 ; resp. 20.
II. 60 minutes <i>after</i> "     "     "     "	Pulse, 82 ; resp. 18.
III. 143 minutes     "     "     "     "	Pulse, 84 ; resp. 20.
IV. 225 minutes     "     "     "     "	Pulse, 79 ; resp. 17.

The pulse-wave, as obtained by the sphygmograph, is not always a very reliable index of the blood-pressure ; yet, as the present observations were made with the greatest care, and with the assistance of Dr. Logan, a former resident physician in the infirmary, and who had considerable experience in the use of the sphygmograph whilst assisting Professor T. R. Fraser, we are entitled to regard these tracings as fairly representing the state of the pulse. In each case, the pressure of the sphygmograph

remaining constant, the instrument was so placed as to obtain the greatest possible movement of the lever, and in this position it was maintained during the experiment. When it was removed to permit of the patient going to stool, as happened in the last two experiments, it was afterwards placed in exactly the same position, and gently moved about until it rested on the point which gave the maximum movement of the lever. In each experiment many more tracings were taken than those reproduced, and all of them showed progressively the changes seen in those selected.

In the first place, it will be observed that the frequency of the pulse cannot be said to be much altered; if there be any change it is towards a slowing of the pulse. It is otherwise with the tension of the pulse, particularly in the first of the three experiments where each successive tracing indicates a progressive increase of the arterial tension, most evident in the third and following tracings. The same effect was observed in the second experiment, although not so markedly. The great difference in the degree of the concentration of the solution of the salt administered in these two experiments producing a remarkable difference in the degree of the concentration of the blood (*vide* Experiments LIV. and LV.) may account for the difference in the pulse. The lessened total volume of the blood in the first experiment is, almost contrary to expectation, associated with a more decided increase of the tension of the pulse. But whether this greater rise is to be attributed to the concentration of the blood, or to the absorption and presence of more salt in the blood than in the second experiment, it is difficult to say.

In the third experiment, that in which sulphate of magnesia was administered, an increase of the tension of the pulse is also apparent. I regret that, owing to the discharge of the other patient from the hospital, I was unable to make this experiment also on him, so that a better comparison might have been instituted between the action of the soda salt and that of the magnesia salt. The frequency of the pulse is in this experiment decidedly lessened, but this may have been accidental.

The rate of the pulse, judging from these and other experiments, becomes usually a little slower after the administration of the salt, but often it is not at all affected.

If I am correct in interpreting the changes observed in the pulse-tracings as indicating an increase of the arterial pressure, it falls to be asked how this increase is accomplished. It is probably due, as I have already hinted, to the irritation of the walls of the arteries, particularly of the smaller arteries, by the salt, which the blood has absorbed, producing a contraction of their calibre, and thus increasing the resistance to the onward flow of the blood. The nature of the alteration of the pulse-wave points more to a diminished outflow of blood through the arterioles than to an increased inflow from stimulation of cardiac action. If the heart participate in effecting the increase of the blood-pressure, the rate of its pulsations shows that it is not by any increase of their frequency.

What then is the value of the cathartic in inflammation, if it increases the blood-pressure within the inflamed organ? Increase of arterial pressure, however, does not necessarily imply increase of pressure within the capillaries, if the increased pressure be due to the contraction of the arterioles. It may, on the contrary, be associated with a diminished capillary pressure; and this is probably what occurs after the administration of a saline cathartic. But, even should the salt increase the pressure of the entire blood system of the organ, it is possible that as much benefit may be derived from a more forcible and penetrating circulation, driving onwards the stagnating blood, as from a diminution of pressure. How else can digitalis prove so beneficial in cases of inflammation and fever, if we accept the results of some recent continental experiments with this remedy?

#### SERIES OF EXPERIMENTS, H.

The effect of the salt on the urinary secretion.

The experiments of this series are, like those of the last series, somewhat fragmentary, and do not pretend to do more than roughly indicate the probable mode of action of the saline purgative on the urine.

This action requires to be considered with reference to the effect of the salt on the rate of the secretion of the urine, and its effect on the chemical composition of the urine. The immediate effect of the administration of a dose of a saline purgative on the



secretion depends considerably on the degree of dilution of the salt given. If the solution is very concentrated, the amount of urine secreted during the next few hours is often somewhat diminished, owing to a partial metastasis of the secretion. But quite as frequently the rate of the secretion may not at all be affected. If, however, the solution is very dilute, there is an immediate increase of the secretion, which continues for a short time, until the excess of water taken with the purgative has been eliminated by the kidneys. But whether the salt solution administered be dilute or concentrated, unless extremely dilute, there is frequently observed, during the twelve hours or so immediately following the injection of the purgative, a diminution of the total amount of the secretion. This diminution is probably dependent on the fluids of the blood having been largely removed during this time by purgation. This effect, however, is not constant, and is not exhibited in all of the experiments selected to illustrate this series. Sooner or later the diminution of the secretion, if it occurs, is succeeded by a generally well-marked increase, which usually begins to be evident within twelve or fifteen hours, or more, after the administration of the purge, and which continues for a complete day, or longer, afterwards. This is well illustrated in Experiment XXIII. of Series A., and in Experiments LIV. and LV. of Series C., and in some experiments whose details I shall immediately give, in all of which care was taken that the daily amount of fluids imbibed was as nearly as possible constant. This diuretic effect of the salt is accompanied by a tolerably distinct concentration of the blood, as I have previously pointed out (Series of Experiments, C.).

The effect of the saline cathartic on the quality or composition of the urine is, apart from the alteration produced by its own presence, not great, and will be best appreciated after considering the results of the subjoined experiments.

In the first of the experiments of this series the action of a very dilute solution of sulphate of soda on the rate of the urinary secretion will be exhibited and contrasted with the normal rate of secretion, and with the effect of water, equal in quantity to the salt solution administered. This, as well as the two subsequent experiments, I conducted on myself; and whilst they were being made I took care that, for some days previously,

my diet, and particularly the liquids of it, should be of the same quality and quantity on each day; as far as possible, also, the amount of physical exercise, and the nature of my occupation, were similarly regulated. Breakfast was taken about 8.30 A.M., and dinner not until 5.30 or 7.30 P.M., no food having been eaten in the interval, and no supper afterwards. No fluids were consumed unless at meals, and then sparingly. In this way it was possible to procure a tolerably steady rate of urinary secretion. The urine was usually collected, by a complete evacuation of the bladder, every half hour during the afternoon, the salt not being administered until about four hours after breakfast, so that the stomach might be fairly free from food, the breakfast being light and easily digestible.

*Experiment CXV.*—In the accompanying tabulated arrangement of the results of this experiment, at each hour bracketed in the first day of the B. part of it, 100 c.c. of a solution of sulphate of soda were drunk, containing 3 grammes of the salt, and at the same hour in the C. part, 100 c.c. of ordinary spring water were taken. The bladder was evacuated every half hour from 12.30 to 7.30 P.M. From 7.30 P.M. until 8.30 next morning it was evacuated at irregular intervals, but the whole of the urine was measured, and from it was estimated the half-hourly average. From 10.30 A.M. to 12.30 P.M. the bladder was emptied hourly. The sulphuric acid of each of the half-hourly evacuations was estimated by boiling the urine with hydrochloric acid, and afterwards precipitating with barium chloride in the usual manner, and with the usual precautions. On account of the urine having been previously boiled with hydrochloric acid, both the “gepaart” and “ungepaart” sulphuric acid was precipitated. The excess represented in the table, and calculated as sulphate of soda, is the excess of the acid due to the presence of the purgative salt. The normal proportion of the acid was estimated during the hour previous to commencing the experiment. The quantity normally excreted very gradually becomes less during the afternoon, but the fall is not sufficient to impair the general accuracy of the results given.

A moderately soft stool was passed about 8 P.M. of the same day on which the salt was taken.

It will be observed from the results of this experiment that the drinking of 400 c.c. of a 3 per cent. solution of sulphate of soda produces an immediate increase in the flow of urine, although not by any means in proportion to the amount of fluid taken, and very much less in amount than that caused by the drinking of the same quantity of water, when, indeed, allowing for the normal amount of secretion, more urine was secreted than there

was taken of water. The immediate increase of the urine, which follows the administration of the salt, gradually but quickly disappears, and is followed by a remote increase, which commences in the course of the night and is apparent during the greater part of the following day.

The rate of the elimination of the salt in the urine is of

*Experiment CXV.*

Time of Evacuation of Bladder.	Half-hourly Rate of Secretion of Urine.				Sulphates estimated as $\text{Na}_2\text{SO}_4 \cdot 10\text{H}_2\text{O}$ recovered from Urine of B.	
	A. Normal.	After Administration of				
		B. Sulphate of Soda.	C. Water.	Total.	Excess.	
						First day.
...	c.c.	c.c.	c.c.	c.c.	grms.	grms.
10.30 A.M.	...	...	...	...	...	...
11.30 "	24	19	29	20	...	...
12.30 P.M.	22	20	32	23	0.1200	...
[12.32 " ]	...	[ 100 c.c. of saltsolution ]	...	[ 100 c.c. of water ]	...	...
1 "	20	32	28	36	0.1989	0.0789
[1.2 " ]	...	[ 100 c.c. of saltsolution ]	...	[ 100 c.c. of water ]	...	..
1.30 "	19	32	30	72	0.1972	0.0772
[1.32 " ]	...	[ 100 c.c. of saltsolution ]	...	[ 100 c.c. of water ]	...	...
2 "	16	48	28.5	172	0.1964	0.0764
[2.2 " ]	...	[ 100 c.c. of saltsolution ]	...	[ 100 c.c. of water ]	...	...
2.30 "	16	51	26	168	0.1968	0.0768
3 "	15	35	25	109	0.2168	0.0968
3.30 "	15	31	25	94	0.2232	0.1032
4 "	14.5	28	26	56	0.2290	0.1090
4.30 "	13	22	24	31	0.2172	0.0972
5 "	13.5	24	23	22	0.2600	0.1400
5.30 "	12	23	17	18	0.2582	0.1382
6 "	11	16	16	13	0.2213	0.1013
6.30 "	11	11	15	12	0.2189	0.0989
7 "	11.5	10	14	11	0.2046	0.0846
7.30 "	11	10	12	10	0.2032	0.0832
10.30 A.M.	28	30	31	25	Total } Excess }	1.3617

particular interest in connection with the experiments of Series D., where the conclusion was arrived at, that, at least, the acid of the salt in large part enters the blood, or rather disappears from the alimentary canal immediately after its ingestion. If it were

present in the blood, we would expect that during this period there would be more of the salt or the acid in the urine than at a later period when the salt returns to the alimentary canal. The rate of the elimination of the salt in the present experiment exhibits no evidence of the blood being richly charged with the salt during the first one or two hours after the administration of the purge.

The next two experiments are similar to the B. part of the preceding in their arrangement, excepting that the salt administered was the sulphate of magnesia, and that the half-hourly excretion of urea and chlorides was carefully ascertained, the former as representing the organic, the latter as representing the inorganic, matter of the urine; the rate, also, of the elimination of the base of the purgative salt, as well as that of the acid, was investigated. In the one experiment a highly-concentrated solution of the salt was taken, in the other a dilute solution, although not so dilute as in Experiment CXV. In the latter of the two experiments the degree of the acidity of the urine was observed. The results of these experiments are arranged in a tabular form.

The urea was estimated by means of the nitrogen or hypobromite process, and the chlorides by the usual volumetric method with nitrate of silver. The magnesia was separated in the form of the ammonio-phosphate, the urine having been previously deprived of its iron and its lime by first acidulating it with hydrochloric acid, then almost neutralising it with carbonate of soda, afterwards adding acetate of soda, and boiling and filtering, and, finally, treating the filtrate with oxalate of ammonia, and, after allowing it to stand for twelve hours, again filtering. The ammonio-phosphate was heated to redness, and the pyro-phosphate obtained, which was the salt weighed. The standard solution of caustic potash employed for the determination of the acidity of the urine contained 0.58 grms. in 100 c.c. of water. As the quantity of the chlorides and urea secreted in a given period depends very largely on the amount of the urine, I have thought it desirable to give the quantity of each in 5 c.c. of urine, as well as the absolute quantity excreted in the course of the half hour. The estimation of the chlorides and of the urea were not continued after dinner, as the food considerably affects their elimination.

The rate of elimination of the chlorides and urea in the normal urine was found to exhibit, as is well known, an increase for a few hours after breakfast, as, indeed, after every meal, and afterwards to undergo with occasional variations a continuous decrease. The decrease was more marked in the case of the chlorides than of the urea.

*Experiment CXVI.*

Time of Evacuation of Bladder.	Quantity of Urine.		Urea.		Chlorides estimated as NaCl.		Sulphates estimated as		Magnesia estimated as	
	At each Eva- cuation.	Rate per half-hour.	In 6 c.c. of Urine.	m. grms.	In 5 c.c.	Per half-hour.	SO <sub>4</sub> . Per half-hour.	MgSO <sub>4</sub> .H <sub>2</sub> O. Per half-hour.	MgO. Per half-hour.	MgSO <sub>4</sub> .H <sub>2</sub> O. Per half-hour.
6.30 P.M.	c.c.	c.c.	m. grms.	m. grms.	m. grms.	m. grms.	grms.	grms.	grms.	grms.
8.30 A.M.	...	...	...	...	...	...	...	...	...	...
[8.45 " ]	640	30	...	...	...	...	...	...	...	...
10.30 "	Breakfast.	20.7	...	...	...	...	...	...	...	...
11.30 "	83	94	389	64	265	...	...	...	...	...
12.30 P.M.	50	25	103	515	75	375	...	...	...	...
[12.32 " ]	54	27	92	496	76	410	0.0374	0.1150	0.0033	0.0205
1	Administered 20 grammes of sulphate of magnesia dissolved in 20 c.c. of water.	31	90	558	79	489	0.0436	0.1341	...	...
1.30 "	23	23	88	403	75	345	0.0436	0.1341	...	...
2 "	21	21	93	390	76	319	0.0606	0.1862	0.0074	0.0458
2.30 "	20	20	96	384	74	296	0.0563	0.1729	...	...
3 "	20	20	98	390	72	288	0.0567	0.1744	...	...
3.30 "	20	20	98	390	72	288	0.0755	0.2321	...	...
4 "	19	19	98	372	72	274	0.0793	0.2440	0.0069	0.0424
4.30 "	18	18	98	352	72	260	...	...	...	...
[5.30 " ]	Dinner.	...	...	...	...	...	...	...	...	...
6.30 "	100	25	86	430	73	365	0.1000	0.3075	0.0064	0.0877
8.30 A.M.	904	32.3	...	...	...	...	0.0880	0.2706	...	...
[8.40 " ]	Breakfast.	...	...	...	...	...	...	...	...	...
12.30 "	196	24.5	...	...	...	...	0.0718	0.2209	0.0051	0.0313
5.30 "	220	27.5	...	...	...	...	0.0632	0.1946	0.0031	0.0191

*Experiment CXVII.*

Time of Evacuation of Bladder.	Quantity of Urine.		Acidity of Urine per half-hour.	Urea.		Chlorides estimated as NaCl.		Sulphates estimated as		Magnesia estimated as	
	At each Evacuation.	Rate per half-hour.		In 5 c.c.	Per half-hour.	In 5 c.c.	Per half-hour.	SO <sub>4</sub> Per half-hour.	MgSO <sub>4</sub> ·7H <sub>2</sub> O Per half-hour.	MgO Per half-hour.	MgSO <sub>4</sub> ·7H <sub>2</sub> O Per half-hour.
10.10 A.M.	c.c. ...	c.c. ...	c.c. ...	m.grms. ...	m.grms. ...	m.grms. ...	m.grms. ...	grms. ...	grms. ...	grms. ...	grms. ...
10.45 "	80	25.7	(alkaline)	...	...	...	...	...	...	...	...
11.15 "	35	35	(acid)	102	720	56	392	0.0361	0.1109	0.0030	0.0184
12.15 P.M.	84	42	4.8	65	551	43	361	0.0355	0.1092	0.0029	0.0181
12.16 "	Administered 15 grammes of sulphate of magnesia, dissolved in 250 c.c. of water.										
12.45 "	90	90	6.5	34	623	21	378	0.0441	0.1357	0.0061	0.0378
1.15 "	90	90	8.5	28	522	16	288	0.0571	0.1756	0.0046	0.0283
1.45 "	29	29	8.0	53	341	35	203	0.0606	0.1862	0.0054	0.0332
2.20 "	25.5	22	9.9	67	298	39	175	0.0629	0.1936	0.0050	0.0307
2.45 "	12.5	15	8.6	50	151	31	93	0.0396	0.1217	0.0047	0.0290
3.15 "	17	17	8.6	60	203	42	143	0.0701	0.2157	0.0046	0.0285
3.45 "	16.5	16.5	8.8	66	218	...	...	0.0584	0.1795	0.0060	0.0371
4.15 "	17	17	9.0	64	218	...	...	0.0556	0.1712	...	...
4.45 "	17.5	17.5	9.7	64	225	...	...	0.0616	0.1894	0.0042	0.0261
5.15 "	15	15	8.6	58	174	41	123	0.0562	0.1729	...	...
6.15 "	31	15.5	8.7	119	368	59	183	0.0673	0.2071	0.0060	0.0371
7.15 "	26.5	13.25	...	119	316	64	169	0.0797	0.2452	...	...

The rate of their elimination did not exhibit a substantial difference from that shown in Experiment CXVI. after the administration of the salt, and, as it varied somewhat from day to day, I have not deemed necessary, for the sake of comparison, to insert the results I obtained.

If a careful survey be made of the results of these two experiments it will be seen, first, as regards the rate of the secretion of the urine, that the administration of the concentrated purge has not been followed by an immediate lowering of the rate, as I have sometimes observed to occur; and that an hour after the diluted salt was taken the secretion has become considerably increased. The further course of the rate of the secretion is much alike in both.

Next, as concerns the acidity of the urine, it becomes distinctly increased after the administration of the salt, but this increase is probably for the most part physiological, and would have taken place although the salt had not been administered, as the urine, which is alkaline whilst gastric digestion is actively going on, gradually becomes acid afterwards, and the salt was probably taken whilst the natural acidity was still on the increase. At any rate, I have several times observed that the normal acidity of the urine, some hours after breakfast, is quite equal to that recorded as occurring after the administration of the salt.

The effect of the salt on the amount of the urea is not well pronounced, and it is extremely difficult to rightly interpret the results obtained, owing to the amount of the urea being greatly affected by the quantity of the urinary secretion. From a careful consideration of these results, and attaching more importance to the percentage than to the absolute amount of urea, particularly where the rate of the urinary secretion has become tolerably uniform, as from 1.30 to 3.30 P.M. in Experiment CXVI., and remembering that the urea normally begins steadily to decrease some hours after a meal and when no exercise is being taken, I am inclined to believe that there is a very slight increase in the excretion of the urea, and that, therefore, the salt promotes to a small extent the tissue-metamorphosis of the body. This is, however, at best, extremely doubtful, and does not receive much support in the next two experiments.

The chlorides of the urine are also evidently not much altered

in quantity, if allowance be made for the gradual diminution in their excretion, which begins after digestion has been completed. If altered they are probably slightly diminished. The estimations of the chlorides wanting in Experiment CXVII. are due to the urine having had hydrochloric acid accidentally added to it.

The most interesting, because, to a large extent, the most definite, of the results of the analyses of the urine in these experiments are those which exhibit the rate of elimination of the acid and base of the purgative salt by the kidneys. This is best shown in the following table, where the excess of the

EXHIBITING THE EXCESS OF THE ACID AND BASE OF THE PURGATIVE SALT ELIMINATED IN THE URINE.

EXPERIMENT CXVI.			EXPERIMENT CXVII.		
Time of Evacuation of Bladder.	Half-hourly Excess, calculated as $\text{MgSO}_4 \cdot 7\text{H}_2\text{O}$ , of		Time of Evacuation of Bladder.	Half-hourly Excess, calculated as $\text{MgSO}_4 \cdot 7\text{H}_2\text{O}$ , of	
	Sulphuric Acid.	Magnesia.		Sulphuric Acid.	Magnesia.
	grms.	grms.		grms.	grms.
12.32 P.M.	Salt administered.		12.16 A.M.	Salt administered.	
1 "	0.0191	0.0253	12.45 "	0.0257	0.0195
1.30 "	0.0191		1.15 "	0.0656	0.0100
2 "	0.0712		1.45 "	0.0762	0.0149
2.30 "	0.0579		2.20 "	0.0836	0.0124
3 "	0.0594	0.0219	2.45 "	0.0117	0.0107
3.30 "	0.1171		3.15 "	0.1057	0.0102
4.30 "	0.1290		3.45 "	0.0695	0.0188
6.30 "	0.1925		4.15 "	0.0612	
8.30 A.M.	0.1556	0.0172	4.45 "	0.0794	0.0078
12.30 P.M.	0.1059	0.0108	5.15 "	0.0629	
5.30 "	0.0795	0.0000	6.15 "	0.0971	0.0188
			7.15 "	0.1352	
Total Excess in 17 hours.	7.3708	0.8074	Total Excess in 7 hours.	1.1061	0.2061

magnesia and the sulphuric acid beyond the quantity normally present in the urine is represented. The normal quantity of each was ascertained from an estimation of its amount in the urine secreted previous to commencing the experiment; and, although the urine examined was collected only during two hours, or a little less, the quantity of magnesia and sulphuric acid obtained from it was found by comparison with analyses of



the total twenty-four hours' urine to represent almost exactly the proportion that these other analyses would have led us to expect.

The rate of the elimination of the acid after it has been fairly established, that is, within an hour, or an hour and a half, after the administration of the salt, remains tolerably uniform for a few hours subsequently, but it gradually increases towards evening, in spite of the quantity of the urinary secretion undergoing a gradual diminution, and remains tolerably high throughout the day. The degree of concentration of the solution of the purgative salt when given, evidently exerts little influence on the rate of the elimination of the acid. The rapidity of the elimination of the base, or the magnesia, was probably also much alike in both experiments, although the results of Experiment CXVI. exhibit a somewhat higher rate than those of the other. The apparent lower rate of the latter is probably due to the errors accompanying the analysis of very small quantities of the urine. In Experiment CXVI., which is more extended than the other, the rate of elimination of the magnesia after the first three or six hours is seen gradually to decline, and terminates evidently some hours previous to the complete elimination of the sulphuric acid. The most noteworthy result, however, is brought out in a comparison of the rates of elimination of the acid and base. The former appears in the urine in much larger quantity than the latter, especially after the first hours following the ingestion of the salt. Estimating both acid and base as  $\text{MgSO}_4 \cdot 7\text{H}_2\text{O}$ , the quantity excreted of the former is on an average nearly ten times as great as that of the latter. The salt is evidently split up somewhere in the body, either in the intestinal canal or in the blood, almost for certain in the former. Assuming that the decomposition occurs within the intestines, and some recent experiments of mine—made in continuation of the experiments of Series D., and since these were published—render it almost a certainty, the base remains for the most part within the canal, and is evacuated along with the faeces, whilst a large proportion of the acid passes into the blood, and is excreted by the kidneys.

A *résumé* of these additional experiments to Series D. has been pub-

lished in a recent number of the *British Medical Journal*,<sup>1</sup> in which it will be observed that, by estimating both the acid and the base of the purgative salt recoverable from the intestinal canal, I have found that the base does not undergo the rapid absorption spoken of in the experiments of Series D., in which the acid of the salt was alone estimated. In these later experiments sulphate of magnesia, as well as sulphate of soda, was employed, whereas in my former experiments the latter salt alone was used. With both salts the results are the same; the magnesia, in the one case, and the soda, in the other, disappears very slowly from the alimentary canal, whilst the acid is rapidly absorbed, and pursues the course already described under Series D. This satisfactorily explains why the sulphate of magnesia does not produce the toxic effect which was seen to result from its injection into the blood. The magnesia never enters the blood in a quantity sufficient to permit of its toxicity becoming developed.

The results of the present experiments are in harmony with those of the experiments I have just alluded to, except that the excessively rapid absorption of the acid which occurs soon after the administration of the salt leaves no trace in the urine of its having taken place; the elimination of the acid is not greater, indeed, is even less, during the first hour following the ingestion of the salt than it is some hours afterwards, when, according to the experiments of Series D., the acid is returning, or has returned, to the alimentary canal, and when, therefore, there ought to be much less of it in the blood, if it be the blood which stores it during its early rapid absorption.

It would be interesting to inquire in what combination the excess of sulphuric acid appears in the urine. As the acidity of the urine is hardly, if at all, greater than normal, it is not in the form of free acid. Most probably it is as a sulphate of the alkaline metals, particularly of sodium.

The next, and the concluding, experiments of this series were made on two convalescent patients in the Infirmary, whose diet was uniform in kind and quantity from day to day throughout each experiment. The daily supply of liquids was also perfectly definite and equal. The object of these experiments was to ascertain the gross and more extended effect of a dose of a purgative salt on the excretion of the urea and chlorides: in the previous experiments the immediate and detailed effect of the salt on the excretion of these substances was determined.

<sup>1</sup> "On the Absorption of Certain Salts from the Alimentary Canal," No. 1146, p. 1204, Dec. 16, 1882.

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*Experiment CXVIII.*—A. W., male, æt. 38. The urine of the twenty-four hours was collected and measured every morning at six, and the purgative (20 grammes of sulphate of magnesia dissolved in half a tumblerful of water) was administered at that hour on the morning of the third day, or at the commencement of the fourth period of twenty-fours, as represented in the following table :—

*Experiment CXVIII.*

Period of 24 hours.	Urine.		Urea.		Chlorides, calculated as NaCl.	
	Total Amount.	Specific Gravity.	In 5 c.c. of Urine.	In total Urine.	In 5 c.c. of Urine.	In total Urine.
I.	c.c. 1426	1014·2	grms. 0·066	grms. 18·836	grms. 0·045	grms. 12·843
II.	1525	1014·5	0·066	20·130	0·046	14·030
III.	1595	1014·3	0·067	21·373	0·042	13·398
	Salt administered.					
IV.	1369	1015·3	0·068	18·618	0·038	10·404
V.	1738	1012·8	0·058	20·160	0·039	13·556
VI.	1025	1014·8	0·064	13·120	0·044	9·020

*Experiment CXIX.*—J. G., male, æt. 47. All the conditions of this experiment were exactly the same as in the previous experiment.

*Experiment CXIX.*

Period of 24 hours.	Urine.		Urea.		Chlorides, calculated as NaCl.	
	Total Amount.	Specific Gravity.	In 5 c.c. of Urine.	In total Urine.	In 5 c.c. of Urine.	In total Urine.
I.	c.c. 1273	...	grms. ...	grms. ...	grms. ...	grms. ...
II.	1267	1017·9	0·078	19·765	0·050	12·670
III.	1353	1016·8	0·084	22·764	0·045	12·195
	Salt administered.					
IV.	1453	1018·0	0·083	24·119	0·037	10·752
V.	1567	1017·7	0·087	27·265	0·049	15·356
VI.	1339	1023·7	0·110	29·458	0·071	19·013

The effect of the purgative on the quantity of the urinary secretion is in accordance with the conclusions derived from my other experiments. The effect on the excretion of the urea is either *nil* or extremely small. The excretion of the chlorides is, on the contrary, considerably modified, being distinctly less during the twenty-fours following the administration of the purgative. The partial disappearance of the chlorides is probably

due to the purgative dejection carrying with it a portion of the chlorides of the body, derived from the blood by means of the intestinal secretion which the cathartic excites.

The experiments of this series are, as I have already said, fragmentary and not numerous enough to warrant definite conclusions, but they render it highly probable that a purgative dose of sulphate of soda or sulphate of magnesia acts as a diuretic as well as a cathartic, the diuretic effect being generally most evident on the day following the administration of the purgative. The action of the salt on the composition of the urine, as tested by the estimation of the urea and the chlorides, is apparently of little moment. Finally, as regards the elimination of the salt itself in the urine, some facts of considerable interest have been elicited, which form a material addition to the results of previous experiments (Series A). Whether the sulphate of soda exhibits the same inequality in the elimination of its acid and base, as the sulphate of magnesia does, I have not yet determined.

*(To be continued.)*

## SOME VARIATIONS IN THE BONES OF THE HUMAN CARPUS. By Professor WILLIAM TURNER, M.B., F.R.S.

No anatomist has done so much to enlarge our knowledge of the variations in the mode of ossification of the carpal bones as Professor Wenzel Gruber, of St. Petersburg. The unrivalled number of subjects at his disposal for purposes of dissection provides him with material for observation on the variations which arise in human structure, such as falls to the lot of no other anatomist. In the *Archiv für Anatomie*, in *Virchow's Archiv*, in the *Bulletin de l'Acad. Imp. de St. Petersbourg*, and in his *Beobachtungen*, he has recorded many examples of modifications in the ossification of the carpus.

This subject has also from time to time attracted my attention, and I have collected several specimens which have occurred in my dissecting-room. The variations which I have met with illustrate diminution in the number of carpal bones through coalescence; increase in number through duplication of the normal bones of the carpus; increase through separation of the styloid process of the third metacarpal bone; increase through the occurrence of a bone which may represent the os centrale in the human carpus.

### *a. Coalescence of Trapezium and Scaphoid.*

In the left carpus of a man one large scapho-trapezium formed the outer part of the carpus. The coalescence was complete, and as there was no mark externally to indicate that the bones might once have been separate, it is probable that in the foetus a single mass of cartilage had represented both these elements of the carpus. The ridge and groove on the trapezial element were distinct, and a rough eminence marked the tuberosity of the scaphoid. The trapezoid fitted into a deep hollow on the inner surface of the conjoined bone. It articulated with the trapezoid, the first metacarpal, os magnum, semilunar, and radius. This carpus possessed only seven bones.

### *b. Division of Semilunar.*

Left carpus of woman. A supernumerary ossicle obviously representing a part of the semilunar was interposed between that

bone and the scaphoid. It measured 0·6 inch from dorsal to palmar surfaces, 0·4 from side to side, and 0·3 from superior to inferior surface. It was wedge-shaped, with the broad end covered with cartilage at the radio-carpal joint, and the narrow end between the semilunar and scaphoid, but not reaching the os magnum. The surface articulating with the semilunar was covered with cartilage, whilst that in contact with the scaphoid was smooth and eburnated. The corresponding surface of the scaphoid and also its radial surface were likewise smooth and eburnated. This carpus possessed, therefore, nine bones.

*c. Division of Trapezoid.*

Right carpus of a man. The trapezoid was divided into a palmar and a dorsal segment, and the dorsal part was about twice as large as the palmar. They both articulated with the second metacarpal below; with the scaphoid above; with the trapezium externally; with the os magnum internally. They also articulated with each other. This carpus also possessed nine bones. This variety is apparently rare, as Gruber seems to be the only anatomist who had previously recorded examples.<sup>1</sup>

In these cases of division of a carpal bone, there can, I think, be little doubt that each segment had been represented originally by a distinct cartilage, and that each of these cartilages had ossified from an independent centre.

*d. The Styloid Process of the Third Metacarpal Bone as a distinct Ossicle.*

It is well known that the carpal end of the third metacarpal is elongated on the radial side of its dorsal aspect into a styloid process, which at times is much more strongly marked than at others. The skeleton of a hand is now lying before me, in which this process projects so far into the carpus as to cut off the base of the second metacarpal from articulation with the os magnum.

In May 1869 Professor Struthers described in this *Journal*<sup>2</sup> an additional bone in the second row of the carpus, which is apparently an example of the styloid process forming a distinct ossicle. In the same year Gruber<sup>3</sup> recorded a case in which the

<sup>1</sup> See his summary in *Beobachtungen aus der Mensch und Vergleich Anat.*, Heft. i. Berlin, 1879.

<sup>2</sup> 1869, vol. iii. p. 354.

<sup>3</sup> *Archiv. f. Anat. Phys. und wiss. Medicin*, 1869, 361, Taf. x. B.

styloid process formed a distinct epiphysis, not united to the end of the third metacarpal. Since then Gruber has described several additional specimens both of uncoalesced epiphyses and of the styloid process forming a separate ossicle.

In the left hand of a man I found, some time ago, on the dorsal aspect of the carpus an ossicle situated immediately at the radial side of the third metacarpal, with which it articulated by a smooth cartilaginous surface, and by a well-marked dorsal ligament. This ossicle corresponded in position to the styloid process. It was wedge-shaped, and measured 0.4 inch from its broad to its narrow end, 0.3 transversely at its base, and 0.45 from above downwards. It fitted between the os magnum and trapezoid, with which it articulated laterally by smooth cartilaginous surfaces, but it did not reach the palmar surface of the wrist. Inferiorly it had, in addition to its joint with the third metacarpal, a slight articulation with the second metacarpal. It is clear that this ossicle had arisen from a centre of ossification separate from the carpal end of the metacarpal bone, and had remained quite distinct. There were, therefore, nine bones in this carpus, but the additional bone represented the styloid portion of the third metacarpal.

*e.* I shall now proceed to describe three specimens, on the exact nature of which there is room for a difference of opinion, as one or other might be regarded either as being a divided scaphoid bone, or as a scaphoid bone along with an os centrale.

*a. Left Carpus of an old man.*—In the interval between the first and second rows of carpal bones was an ossicle half an inch in its transverse, by 0.3 in its dorsi-palmar, and 0.2 in its supero-inferior diameter. It was visible on the dorsal surface of the carpus, but not on the palmar, for it lay between the scaphoid, os magnum, and trapezoid, and was concealed by them in front. It articulated superiorly with the scaphoid, and was, as it were, fitted into a hollow in that bone, the dorsal outline of which was only complete when this ossicle was in its place; the surfaces in apposition were in part smooth and cartilaginous, and in part slightly irregular. Inferiorly it had a smooth articulation with a facet on the trapezoid, distinct from the facet on the same bone for the scaphoid; whilst internally it contributed slightly to the formation of the boat-shaped hollow of the scaphoid, in which

the head of the os magnum is lodged. Its dorsal surface was attached to the dorsum of the scaphoid by a ligamentous band. The scaphoid itself was about the usual size, and possessed a well-marked tubercle.

*β. Left Hand of a man.*—The scaphoid was much diminished in size, and modified in shape. Its transverse diameter was only 0·8 inch, and its dorsi-palmar 0·9. It was almost entirely concealed on the palmar aspect of the carpus between the radius and an additional bone to be described immediately; it appeared on the dorsum of the carpus, where it formed a narrow surface, rough for the attachment of ligaments; it articulated with the radius above, with the semilunar and os magnum internally, and with the additional bone below, its surface for the last named being smooth, cartilaginous, and elevated into a ridge, which fitted into a corresponding hollow on the superior surface of the additional bone. That portion of a normal scaphoid which is prolonged into the tubercle was not represented in this bone.

The additional bone was larger than the scaphoid as just described; it was 1·1 inch in its transverse, the same in its greatest dorsi-palmar diameter, and 0·4 in its greatest supero-inferior. It articulated with the os magnum internally; with the trapezium and trapezoid below; principally with the scaphoid above, but slightly at its inner border with the semilunar, and somewhat more at its outer border with the radius, which was grooved immediately internal to the styloid process to receive it. As the outer border, extended radiad to the scaphoid, the external lateral ligaments of the wrist were attached to it. The palmar surface was rough for attachment of ligaments, and was not prolonged into a tubercle. The dorsal surface was also rough, and attached to it by a ligament was an irregular ossicle, 1·3 inch long and 0·6 broad, which lay transversely on the back of the carpus, and had probably been formed by an ossification in the dorsal ligaments.

*γ. Left Hand of a woman.*—The scaphoid was about one-half the natural size, and represented in its shape only the inner half of the bone. Had the radius been preserved, I have no doubt that one would have seen it concealed on the palmar aspect between that bone and an additional bone to be described immediately. The scaphoid appeared on the dorsum of the carpus, where it



possessed a surface, broader than in specimen  $\beta$ , for the attachment of ligaments. It articulated with the radius above; with the semilunar and os magnum internally, and with the additional bone below, its surface for the last named being convex and covered with cartilage. The part of the scaphoid which is prolonged into a tubercle was not represented in this bone.

The additional bone was larger than the scaphoid as just described; it was 0.6 inch in its transverse, 0.7 in its greatest dorsi-palmar, and 0.3 in its greatest supero-inferior diameter. It articulated with the os magnum internally; with the trapezium and trapezoid below, and with the scaphoid superiorly. In the absence of the radius, I cannot say definitely if it had an articulation with that bone, but I am inclined to think that it had not; it did not articulate with the semilunar. It appeared on the palmar, dorsal, and external surfaces of the carpus, and its surfaces there were all rough for ligaments. As it extended radiad of the scaphoid, the external lateral ligaments of the joint were attached to its outer surface. Its palmar surface was not elongated into a tubercle like the tubercle of the scaphoid.

Lying on the dorsal surface of the carpus, and connected by ligament with the dorsum of this additional bone, was a slender ossicle 0.6 inch long; to the opposite end of this ossicle a more minute nodule 0.2 inch long was attached by ligament. They had apparently been developed in the dorsal ligaments of the wrist.

The additional bone in  $\alpha$  differs in its shape, size, position, and relation from the additional bones in  $\beta$  and  $\gamma$ . In  $\alpha$  it was situated between the first and second rows, and articulated with the scaphoid, os magnum, and trapezoid, but not with the trapezium. In its position and articulations therefore it corresponded with the os centrale in the carpus of the Orang, except in its want of an articulation with the trapezium. But it was very much smaller than the Orang's os centrale, which in the adult measures about 0.9 inch in its longest diameter. It had the appearance of a segment cut out of the scaphoid, but ossified independently of it; but this indeed is not unlike the aspect of the Orang's os centrale, which is received into a hollow on the inferior surface of the scaphoid. There seems reason, therefore, to regard this ossicle, notwithstanding its small size, as representing an os centrale.

In  $\beta$  and  $\gamma$  the "additional bone" although differing in some features of detail, yet obviously correspond with each other in their position and chief relations. The questions to be considered in regard to them are—whether they are to be regarded as an os centrale in each carpus, or as due to a division of the scaphoid into two parts. If the latter, then the bone, which I have named scaphoid, would correspond to that called by Gruber<sup>1</sup> naviculare secundarium ulnare; whilst the additional bone would be his naviculare secundarium radiale. There is much to be said in favour of this latter view. For the bone described by me as the scaphoid only represents a portion of that bone, viz., the part which articulates with the semilunar, i.e., the ulnar portion. Again, the "additional bone" extends radiad of the more internal element, forms a part of the outer border of the wrist, and in one instance at least articulates with the radius. Further, in the carpus of an Orang, and in that of a large Baboon, with which I have compared these specimens, the scaphoid bone—co-existing along with an os centrale—possesses the characteristic shape, is not diminished in size in its transverse diameter, and is elongated into a well-defined tubercle at the outer part of its palmar surface. Hence, the co-existence of an os centrale, along with a scaphoid, in these apes, does not affect the proper scaphoid characters; whereas in  $\beta$  and  $\gamma$  if the additional bone be an os centrale, then the scaphoid is reduced to its inner or semilunar half only. On the other hand, it must be stated that if the "additional bone" is a naviculare secundarium radiale, then it is not shaped at all like the outer half of the scaphoid bone. It is larger, almost of equal diameter in its transverse and dorsi-palmar diameters, so that it has a plate-like character, and it has no tubercle on the palmar surface.

<sup>1</sup> *Archiv f. Anat. und Phys.*, 1866, § 365; *Virchow's Archiv.*, 1877, Bd. 69.

**MULTIPLE RENAL ARTERIES.** By A. MACALISTER, F.R.S.,  
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IRREGULARITIES of the renal arteries are the commonest varieties met with among the abdominal vessels ; indeed, these arteries present some form of variation in three cases out of every seven.

Leaving<sup>1</sup> out of account those varieties which are associated with misplaced or horse-shoe kidneys, we may classify the many forms of anomalous renal arteries as follows :—

1. *Varieties of Numbers.*—The arteries may be—(a) diminished in number, and this under two conditions—(a), with absence of the left kidney, as in Weissman's case ; or (b), with the origin of both renals from a common stem arising from the front of the aorta, as in Portal's well-known instance. Very much more commonly (β) the arteries are increased in number.

Multiple renal arteries may be threefold—(a), most commonly the additional branches spring from the aorta ; (b), or they may come from other sources ; or (c) there may be a co-existence of additional vessels from both sources.

Of the first class, there have been described cases of—

$$\left. \begin{array}{l} \text{one,} \\ \text{two,} \\ \text{or} \\ \text{three} \end{array} \right\} \begin{array}{l} \text{right aortic renals} \\ \text{associated with} \end{array} \left\{ \begin{array}{l} \text{one,} \\ \text{two,} \\ \text{three} \\ \text{or} \\ \text{four} \end{array} \right\} \begin{array}{l} \text{left aortic renals.} \end{array}$$

Of these twelve varieties, I have not found the variety of two right and four left, and I have seen, in addition, single instances of three right and five left, and three right and six left. The commonest form, next to the normal condition of one on each side, is two on the right and one on the left. The second commonest condition is the reverse ; but among the forms with larger numbers the greatest number is more frequently seen on the left than on the right side. In all these cases one vessel arises in the position of the normal renal ; a second commonly springs from the aorta much lower down, generally on the level of, or below, the inferior mesenteric ; the third, when present, is a very short distance above the normal renal, very close to the supra-renal, and on

<sup>1</sup> In cases like those described by Hunter (*Med. Trans. of the London Coll. of Physicians*, vol. iii., 1785, p. 250) and by John Reid (*Phys. Path. and Anat. Researches*, p. 417). Where there were two kidneys on the right and none on the left, there were two right renal arteries—an upper and a lower—and none on the left. For other cases of deficient simple kidney see Professor Watson's Paper (*Ed. Med. Journal*, 1874); and several are recorded in the *Trans. of the Path. Soc.*

the level of the superior mesenteric (this branch is not to be confounded with the form, to be hereafter noticed, of a renal branch from the supra-renal). These multiple branches have been described by most anatomists, so I need not give references. Cases of five on the right are described by Otto and Meckel, and other multiple forms are recorded by many of the older anatomists.

2. *Varieties of Origin*.—Additional renals often spring from other sources, in the following order of frequency:—(α), the supra-renal, a very common source of an upper renal artery; (β), the second, or (γ), the third lumbar artery; (δ), the right hepatic; (ε), the colica dextra; (ζ), the external iliac; (η), the internal, or (θ), the common iliac; or (ι), from the middle sacral. Of all but the first I have seen but single instances. Parallel cases, however, are quoted by Otto, who has seen two instances of the last form, where the anomalous branch went respectively to the right and to the left kidneys. Otto also records a curious and unique example, in which a branch from the right common iliac supplied the left kidney.

The most remarkable instance of this class of variety which I have noted is one which I have preserved in our University Anatomical Museum, taken from a male adult subject.

In this case, on the right side, there are three renals, two from the aorta—a normal, and an inferior—and one from the capsular artery. There is no capsular branch of the aortic renal on this side. On the left side there are six renals from the following varied sources:—three from the trunk of the abdominal aorta, a normal, an inferior, and a superior, which arises directly below the left aortic supra-renal, and sends an inferior capsular branch to that organ, and enters the superior extremity of the kidney. The normal renal bifurcates before it reaches the hilus.

A fourth renal artery springs from the front of the aorta, immediately above its bifurcation, and with its origin a little to the right of the middle line. If this origin were a quarter of an inch lower it would be comparable with Otto's otherwise singular instance above quoted. The fifth renal arises from the sacra media, about half an inch below the origin of that vessel, crosses over the left common iliac artery underlying the ureter, and entering the lower part of the hilus of the kidney. The sixth and lowest branch arises from the internal iliac artery immediately at its point of division, ascends, crosses the common iliac, and pierces into the lower part of the gland. This instance is thus remarkable as combining in itself three of the rarest forms of anomaly hitherto described.

3. *Anomalies of branching* of the renals are very common; indeed, the number of branches whereby the normal renals enter the substance of the kidney is very inconstant; three or four are the commonest numbers, but I have seen up to ten penetrating branches. I have, however, preserved no record of the relative frequency of these. Otto describes the renals in one case as branching into very many branches. The other extreme, that is, the entrance of the renal by a single branch into the glandular substance, is rarer than multiple division.

4. *Varieties of Entrance.*—The places where renal arteries enter the kidney vary. Usually—(a), all enter at the hilum; (β), one often enters at the lower end, and this in most cases comes from the aorta, but may be a branch of the normal renal, once from the lumbar; (γ), one may enter at the upper end, most commonly a branch of the supra-renal, but which may be from the aorta or normal renal. I have seen a vessel piercing into the front surface of the gland from the normal renal; and in another case a posterior branch passed under the inner edge of the gland and entered the gland at the middle of its posterior surface.

5. *Varieties of Distribution of Branches.*—From the renal there may arise branches—(a), to the supra-renal capsule, very common if not normal; (b), to the diaphragm, once; (c), to the right crus of the diaphragm; (d), to the right colon; (e), to the pancreas, deep surface of the head; (f), to the testis, supplanting the normal spermatic; (g), to the right lobe of the liver. These anomalous branches, with single examples of which I have met, were all on the right side, which is curious, as the majority of the anomalies in Class No. 2 were sinistral.

In connection with these anomalies, it is interesting that in one case of Oppolzer's anomaly, a floating kidney which I dissected, where the organ was almost entirely surrounded with peritoneum, the vessels were normal, as in the case described by Urag (*Wiener Medicinische Wochenschrift*, 857, No. 42).

Multiplication of renal arteries is not surprising when we consider the arrangement of these vessels in other animals and their development. Thus for the elongated kidneys in fishes the arteries are numerous, and with a trace of metamerism in their succession. In the iguana and monitor, among lizards, they are also multiple, as also in snakes. The alligator and crocodile have three or four on each side. Most birds have four, five, or six pairs, of which the three uppermost arise from the aorta, and the two or three lowest from the ischiatic.

The mammalian kidney is the metanephros, or hinder part of the primitive excretory organ, and it originates from a rounded mass of mesoblast, from the intermediate cell mass at that region where the dorsal outgrowth from the Wolffian duct extends forward to the tissue behind and nearer the spine than the rest of the nephros. In this tissue the vessels originate *in situ* in the mesoblast, close to those which supply the abdominal wall. These vessels, which are thus close together, separate at an early period, though traces of this primitive relation persist in the extra-peritoneal anastomoses of the renal arteries, through their arteriæ adiposæ and capsular branches. We owe many of the anomalies above described to persistent accidental enlargements of some of these vessels.

DIVISION OF THE SCAPHOID BONE OF THE CARPUS,  
WITH NOTES ON OTHER VARIETIES OF THE CARPAL  
BONES. By R. J. ANDERSON, M.A., M.D., *Demonstrator of  
Anatomy, Queen's College, Belfast.*

IN a carpus of the left side of a male subject, a division of the scaphoid occurs. The other carpal bones present no unusual features.

The scaphoid consists of two parts, an anterior and a posterior portion. The posterior part is the larger, and has an upper convex surface for articulation with the radius. This surface is eburnated along the inner half of its extent, and the outer half is rough and non-articular; the whole surface measures one inch, and is rather more than a line broad. The inferior surface (outer) articulates with the trapezoid and the trapezium. The outer end is rough and non-articular; the inner end has a slight notch which receives the semilunar. The anterior surface is concave, eburnated, and has grooves from friction; it is broader externally than internally, and articulates with the anterior portion. The internal surface (for os magnum) is narrow, in part articular for the os magnum. The anterior segment is not unlike the pisiform in shape and size. It has an upper convex articular surface for the radius covered with cartilage. A lower concave articular facet which, with the scaphoid, receives the head of the os magnum. The posterior surface is convex, eburnated, and grooved, and articulates with the posterior segment. The inner end articulates with the semilunar.

The abnormal character of the carpus above described resembles those mentioned by Gruber in his various papers, and in position the os centrale of the typical carpus. Professor Gruber points out in one of his papers<sup>1</sup> that a small notch is present in  $\frac{2}{3}$ ths of all the scaphoids, below the ulnar end of its dorsal surface. The notch is generally semilunar, of variable size and depth. This notch is sometimes large, and may receive a ninth carpal bone, which there lies between the trapezoid, the scaphoid, and the os magnum. Professor Gruber brings forward good reasons for believing this to be the representative of the central bone of the carpus. Amongst other English anatomists Professor Struthers found a bone in the situation mentioned by Gruber, and described it in the *Journal of Anatomy and Physiology*.<sup>2</sup> This anatomist considers it to be a fracture with formation of a false

<sup>1</sup> Reichert and Du Bois Reymond's *Archiv.*, 1873, p. 716.

<sup>2</sup> Nov. 1873, p. 113.

joint. In Virchow's *Archiv*,<sup>1</sup> Professor Gruber discusses the question, and points out that, apart from the presence of the small bones in the situations above mentioned, there is nothing that indicates any abnormality due to disease or injury, and states his conclusions thus :—

- (1.) Fractur des Naviculare Carpi geht immer in sagittaler, d. i. in der die Längsaxe des Knochens kreuzenden Richtung und fast an derselben Stelle vor sich.
- (2.) Totale Fractur bestehe immer ohne Dislocation der fragmente.
- (3.) Gelenkknorpel und Articulationen seien selbst bei totaler Fractur so wenig in Mitleidenschaft gezogen, dass Zeichen davon ersteren in späterer Zeit an der Leiche nicht mehr nachzuweisen sein.

Now, in the above-mentioned case, we have evidence of pressure not only between the pieces of the scaphoid (the scaphoid and centrale), but further evidence of pressure and friction at the upper surface of the posterior bone. Whilst in the lower piece the posterior surface is eburnated and worn a groove by the rubbing, whilst the radial surface is covered with normal cartilage. If this example be regarded as a case of the scaphoid and centrale being present, then it remains to explain the cause of the eburnation in two distinct places, whilst all the other parts are free. On the other hand, if it be regarded as an actual fracture, it must then be concluded that fracture of the scaphoid may take place in a direction *parallel* to its longitudinal axis.

#### *Union of the Os Magnum and Trapezoid.*

The bones of the carpus of a right upper extremity present generally no unusual features. The os magnum, however, which has its inner surface normal, and an upper part or head of the usual size and shape, has the piece of bone representing the trapezoid attached to its external surface. Posteriorly the surface of the os magnum is continuous into that of the trapezoid without intermission, but a slight very superficial groove seems to indicate the line of original separation. The upper surface of the trapezoid shows the usual quadrilateral facet, and it is continued into the os magnum without any line marking a division. The inferior surface of the bones is articular for the second metatarsal and third; posteriorly there is no line of separation, but a fissure exists at the anterior part, which occupies the lower and anterior surfaces.

This condition seems to be rare, and the fact that no other carpal bones are involved, leads to the hypothesis that the union took place at a very early period, or that the bones never became completely differentiated. To find the scaphoid and semilunar united, we must look down low in the mammalian series. In certain of the Ruminantia, viz., in Pecora and Tragulina, the trapezoid and os magnum are confluent. In the three-toed sloth the same condition obtains.<sup>2</sup>

<sup>1</sup> Vol. 69, 1877, p. 391.

<sup>2</sup> Flower, *Osteology of Mammalia*.

*Coalescence of the Semilunar and Cuneiform.*

The specimen which I have represents a case of perfect union of the semilunar and cuneiform. The upper surface of the former passes into that of the latter without any interruption, and the large convex quadrilateral facet would seem to limit the extent of the former bone. The anterior faces of the bones are continuous, as also the posterior. The bones can be best limited on the inferior surface. From without inwards first comes an antero-posterior concave facet, then a linear facet for the unciform separated from the former by a ridge, then a groove succeeded by a double curved surface. This condition approaches that of the carpus of *Pteropus*, when the bones of the first row are united. Wagner met with a case similar to that described.<sup>1</sup>

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A FIRST DORSAL VERTEBRA, WITH A FORAMEN AT THE ROOT OF THE TRANSVERSE PROCESS. By Professor WILLIAM TURNER, M.B., F.R.S.

SOME months ago, one of my pupils, Mr. E. J. B. du Moulin, showed me a spinal column which he had purchased from M. Trammond, of Paris, that presented a remarkable variation in the first dorsal vertebra. He has very kindly handed the specimen over to me for the Anatomical Museum of the University.

The first dorsal vertebra had on the left side a foramen through the root of the transverse process sufficiently large to admit an ordinary-sized steel knitting needle. The foramen was immediately anterior to the articular process, and had a vertical direction. It was situated in the pedicle of the vertebra, which was widened antero-posteriorly to accommodate it. External to the foramen the transverse process had the usual tuberculated form, and possessed anteriorly a smooth facet for articulation with the tubercle of the rib. The head of the first left rib, and half the head of the second rib, articulated with the side of the body of this vertebra in the usual way.

On the right side a foramen had also evidently existed in the recent state, but in the macerated bone it was only a deep crescentic notch in the pedicle. The anterior boundary was only imperfectly formed by bone, but without doubt a fibrous band had been connected during life to the horns of the crescentic notch, so as to complete the foramen. The first and second right ribs articulated in the usual way with the right side of this vertebra. In the absence of the soft parts it is, of course, impossible to say what these foramina had transmitted, but in all probability either a vein or an artery, or perhaps both.

<sup>1</sup> Heusinger's *Zeitschrift*, t. iii. p. 300.



The other dorsal vertebræ presented no special peculiarities. The seven cervical vertebræ each possessed a foramen at the root of the transverse process. In the fifth, sixth, and seventh on the right side, and in the sixth on the left side, the vertebrarterial foramen was divided into two portions by a slender bar of bone.

The question now arises, What is the nature of this foramen in the first dorsal vertebra? The vertebrarterial foramen in a cervical vertebra is a hole situated between the anterior division of the transverse process, which is regarded as representing a cervical rib, and the posterior division, which is regarded as the proper transverse process. This view of its position is borne out by what one sees in specimens where cervical ribs are formed in connection with the seventh cervical vertebra. In these cases the anterior division of the transverse process develops into a movable rib, and the interval, between the neck of that rib and the proper transverse process, is regarded as homologous with the vertebrarterial foramen, for there is no other aperture or foramen in a vertebra, so modified, which can be considered as its representative.

In the first dorsal vertebra, now described, in addition to the foramen in the pedicle, there is, in the usual manner, an interval of separation between the neck of the first rib and the front of the pedicle. This interval would, if a cervical rib had been present in this person, have been in series with the interval between that rib and the posterior or proper transverse process of the seventh cervical vertebra, and also with the vertebrarterial foramina in the higher cervical vertebræ. The foramen in my specimen cannot therefore, I conclude, be regarded as homologous with the vertebrarterial foramen in a cervical vertebra, but seems to be a foramen in the pedicle itself, and one, therefore, which has arisen specially in this vertebra as a local and individual peculiarity.

This peculiar condition of the first dorsal vertebra I have seen for the first time in this specimen. It is obviously very rare. In the elaborate article "On Variations of the Vertebræ and Ribs in Man," published in the *Journal of Anatomy and Physiology*, November 1874, Professor Struthers of Aberdeen makes no mention of this variation, and I have not observed any description of it elsewhere in anatomical literature.

# Journal of Anatomy and Physiology.

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## CONTRIBUTION TO THE STUDY OF SPINA BIFIDA, ENCEPHALOCELE, AND ANENCEPHALUS. By Professor CLELAND, F.R.S., *Glasgow*. (PLATES XI. and XII.)

NOTWITHSTANDING all that has been written on such malformations as are included under the terms spina bifida, encephalocele, and anencephalus, and the recognition in a general way of their dependence on dropsy, or on deficient closure of the canal of the cerebro-spinal axis or its coverings, there yet remains room for a more detailed account of their anatomy, and a more explicit comprehension of their nature. This impression is not removed by reference to the recently published work, with Atlas, compiled from many writers by Ahlfeld,<sup>1</sup> nor even by perusal of the instructive pages of Förster.<sup>2</sup> I therefore make no apology for describing a few specimens with more or less full detail.

I. *Hydrocephalus and Spina Bifida in an infant born at full time.*—*Skeleton.*—The dried skull is about  $5\frac{1}{4}$  inches long,  $4\frac{3}{4}$  high, and  $3\frac{1}{4}$  broad. The parietal bones exhibit deep notches and circular perforations at the upper and back parts; and on the upper border of the right parietal some ossa triquetra convert four deep notches into foramina. The frontal bones are much perforated at the lower parts of their frontal plates and anterior parts of their orbital; the orbital plates being completely separate, and other smaller ossifications intercalated between them and the frontal plates. The foramen magnum and cervical vertebræ are normal; but from the sixth dorsal downwards the spinal canal is open, the laminæ being spread

<sup>1</sup> Ahlfeld, *Missbildungen des Menschen*, Leipzig, 1882.

<sup>2</sup> Förster, *Missbildungen des Menschen*, Jena, 1861.

out to the sides in the lower dorsal and the lumbar regions; each lamina touching the lamina below it, and the last dorsal and upper lumbar laminae being greatly expanded (fig. 5). The 3rd and 4th ribs of the left side are fused in the whole extent of their heads and necks and for a quarter of an inch further forwards; and the corresponding vertebral laminae are likewise fused, leaving only a perforation between their united transverse processes. Also, the body of the 4th dorsal vertebra presents in front the appearance of two centres of ossification, the left one the larger. The left laminae of the upper six dorsal vertebrae are more expanded than in the normal foetus, and than the corresponding structures on the right side, and the first of them seems to be supplemented by an additional centre of ossification projecting behind and below it. On the right side the laminae of the 3rd and 4th dorsal vertebrae are normal; but an additional lamina without pedicle or transverse process is intercalated between the 2nd and 3rd. In the sacral region, only the 2nd and 3rd laminae turn inwards towards the middle line, and they are expanded. *Integument*.—In the first place may be mentioned a post-anal dimple bound down to the tip of the coccyx. In the middle third, between this and the head, is an oval space with a smooth thin covering, different from the surrounding integument, and presenting the well-known character of spina bifida, while at the upper end of this oval space or membranous area a minute aperture can be detected. *Cerebro-spinal axis*.—For purposes of exhibition, the spinal cord was divided  $1\frac{1}{2}$  inches below the pons Varolii, and the lower part was removed in connection with the integument of the back, while the upper part was kept in connection with the encephalon. The corpus callosum and fornix had been destroyed by the distending effusion, while the hemispheres were in a state unfit for preservation. The optic thalami and corpora striata were normal, but the corpora quadrigemina were curiously altered in shape, the testes being projected above the nates, and the nates being flattened, probably by the pressure of the distended hemispheres. The cerebellum has its two lateral lobes completely separated by the vallecule; and in it the inferior vermiform process extends up so far, that what appears to be the pyramid touches the corpora quadrigemina, while the uvula looks back-

wards, and the laminated tubercle hangs down from an exaggerated velum posticum, as an appendix  $\frac{3}{4}$ -inch in length lying in the prolonged 4th ventricle (fig. 6). The 4th ventricle extends to half an inch above where the spinal cord has been divided, and there presents an apparently imperforate depression at the end of the central canal of the spinal cord. But at the place where cut across, the central canal was dilated so much that it would admit a probe, and was traced up to within a quarter of an inch from the depression in the 4th ventricle. Turning now to the specimen prepared to show the remainder of the spinal cord and its connection with the integuments, we find that the perforation seen at the upper end of the superficial oval space is continued up into a dilated central canal, and that the cord terminates abruptly here. But division of the dura mater down the middle line in front of the cord has brought into view the continuation of the arachnoid space down to the lower end of the sacral canal, and the nerves on each side given off in order, and clothed with arachnoid. Each is provided with its ganglion outside the dura mater, and appears quite normal. Each root is traceable upwards and inwards, the anterior roots to near the middle line, and the posterior roots to a line on each side; and both are firmly connected with the superficial membrane. By dividing the posterior part of the dura mater on each side, behind the emergence of the nerves, what may be termed the posterior sac of the arachnoid is seen to bifurcate inferiorly opposite the termination of the cord. By slitting up the arachnoid in front, a large subarachnoid space has been brought into view, and a large anterior spinal artery, extending down at least as far as the middle of the superficial membrane of the spina bifida to the level where the 2nd sacral nerves take origin. A delicate layer of the superficial membrane has crumbled away in the manipulations to which the specimen has been subjected, and was no doubt the continuation of the grey matter of the cord, or the epithelium lining the central canal. The tougher layer on which this has rested is to be regarded as the representative of the pia mater and reticulum.

II. *Cervical Spina Bifida*.<sup>1</sup>—The face and scalp are like those of

<sup>1</sup> Presented by Dr. Murdoch Cameron, to whose kindness I am also indebted for others of the specimens described.

an infant at full time ; but, judging from examination of the skull and from the limbs, I should think it is only an eight months' foetus. It is female ; the back is short, the hairy scalp reaching to within 3 inches of the nates. A large fluctuating tumour was felt extending over the sides and back of the head, and down the back. The swelling came forwards to the ears, which are much misshapen, apparently in consequence of pressure. On cutting open the tumour, a larger mesial cyst was found, with an elongation of the 4th ventricle of the brain lying in the bottom of it. At the sides, some minor collections of fluid were separated by thin partitions from the main cavity. *Skeleton*.—The skull does not differ from a normal eight months' skull, except that the parietal and occipital regions are less developed, giving the shape normal at an earlier period. The palate is cleft, but the intermaxillaries fully developed. The twelve pairs of ribs are all present, the first pair having normal sternal attachments. The ribs are crowded together, elongated forwards, and bent inwards, so as to compress the thorax laterally. There are six lumbar vertebræ of normal appearance, and only six cervical vertebræ. The dorsal and cervical vertebræ are much altered ; those to which the five lower pairs of ribs are attached have the laminæ directed outwards and expanded, with the centrum not deviating from the normal save in being broader ; while the vertebræ above these have not only their laminæ directed transversely outwards, but a mesial gap completely separating the right and left moieties of their bodies ; and this gap extends into the upper part of the neck (fig. 2). The cervical vertebræ are greatly shortened and altered. The body of the axis is in two lateral parts, while no separate ossification seems to exist for the odontoid process. Below this, on each side, is a series of three half centra becoming fused together ; then follows on the left side a block of six such, and on the right side blocks of two and four in succession, all much crowded together. The right laminæ of the 2nd, 3rd, 4th, and 5th cervical vertebræ are united externally into one lateral projection, and similarly the 3rd, 4th and 5th of the left side are united. *Cerebro-spinal axis*.—The cerebrum and cerebellum appeared normal ; but the distance from the under surface of the cerebellum to the extremity of the spinal cord is only  $2\frac{1}{2}$  inches, of which  $1\frac{1}{4}$  inch is open behind, so as

to form an elongation of the floor of the 4th ventricle. In the middle of this there is a transverse fold or pucker forwards corresponding in position with the mesial fissure seen in the cervical part of the column, and from this part nerves come off very crowdedly (fig. 7).

III. *Occipital Encephalocele*.—This specimen is of a kind well known in its general characters, having the greater part of the brain projecting behind the skull in the form of a chignon. It was named by the elder Geoffroy Saint-Hilaire "Notencephale," and the skull was carefully figured by him.<sup>1</sup> Rising upwards and backwards from the head is an enormous tumour formed by the brain; it is of such size that it measures about 5 inches across and nearly 3 inches from its uppermost part to the chin. *Brain* (fig. 1).—Only the foremost part of each hemisphere, a portion of the size of a small walnut, was within the cranium, while the rest projected behind; the hemispheres being constricted slightly opposite the enlarged foramen magnum, which formed a circle  $1\frac{1}{2}$  inch in diameter. In front of each hemisphere, occupying the very foremost part of the cranial cavity, was a little cyst about  $\frac{3}{8}$ -inch diameter, with thin walls, which were torn into on removing the brain. These were probably dropsical olfactory bulbs. The interior of the hemispheres was in a condition unfit for dissection, but the surface was copiously convoluted. Their united mass exhibited a bilobate form due to the greater resistance offered by the falx cerebri than by the lateral parts of the dura mater to their expansion backwards. The positions of the origins of the 5th and 8th nerves were so far displaced backwards that these nerves ran a course of nearly three inches forwards before piercing the dura mater. The cervical nerves are all crowded together like the corresponding vertebræ, and the part of the cord from which they spring has maintained its position within the spinal canal, while the medulla oblongata has been so pulled backwards by the displacement of the cerebrum that it is quite unrecognisable; only, it appears from the relative positions of the 5th and 8th nerves that the upper part of the pons Varolii has been the portion of the base of the brain most thrown backwards, and that the part of the medulla

<sup>1</sup> *Mémoires de l'Académie des Sciences*, Mémoire lu en Octobre 1820; and repeated in the *Philosophie Anatomique*.

between the 8th cranial and the 1st cervical is the part stretched. In front of this stretched part lies an elongated basilar artery. The cerebellum is barely recognisable in two lateral parts sundered one from the other, much compressed, and carried backwards beneath the hemispheres. Projecting forwards in the middle line between the elongated nerves, and arising from the part of the base driven farthest back, is a hollow pouch about 2 inches long and  $\frac{3}{4}$ -inch broad, with tolerably tenacious walls, like the finger of a glove. This is the infundibulum and lamina cinerea, and possibly includes the cerebral part of the pituitary body. At the tip of this pouch, in front, one can recognise stretched remains of the optic nerves and some slight vestige of optic commissure. *Skeleton*.—The highest vertebra with laminae united is the 2nd dorsal. The neck is short, and the bodies of the cervical vertebræ show evidence posteriorly of having been each ossified from twin centres. Their laminae remain wide apart from the middle line, and on the right side those of the 2nd and 3rd are completely fused behind their transverse processes, as also are those of the 4th, 5th, and 6th; while, on the left side, those of the 2nd, 3rd, and 4th are similarly fused, and those of the 5th and 6th. The base of the skull, as seen from within, has the petrous bones so bent downwards that their upper borders enclose between them superiorly an angle of  $220^\circ$ ; and it is so curved mesially that the clivus is at right angles to the united bases of the orbital wings of the sphenoid. The frontals are flat above, with their frontal plates pressed into contact with their orbital plates, and prolonged only a  $\frac{1}{4}$ -inch farther back. On the right side the parietal is represented by two bones, an anterior of small dimensions articulating with the frontal and squamous, and a posterior much larger quadrate bone articulating with the mastoid and occipital, and with nearly the whole length of the left parietal. On the left side two corresponding elements can be distinguished, but are ankylosed. The supra-occipital bounds the enlarged foramen magnum in its dorsal moiety, and is only about  $\frac{1}{4}$ -inch broad in the middle line, but is thick and strong. The palate is cleft in its whole length save in the intermaxillary part; and the vomer articulates with a pair of enlarged mesial palatine processes of the intermaxillaries similar to those found in many mammals (fig. 3).

IV. *Occipital Encephalocele, Open Spina Bifida, and Displacement of Viscera*.—This specimen is a female foetus of five months, with a large and solid cerebral chignon of the size of the rest of the head; and beneath this the spinal canal open in its whole length.

*Skeleton*.—The skull<sup>1</sup> differs from that of the encephalocele already described, in that the two lateral divisions of the occipital bone fail to meet in the middle line behind the parietals; but the vault is collapsed in the same fashion. Beneath the skull behind there is a deep recess, in which the membranes of the cord were carried forwards; and in dissecting them out they were found to cohere to viscera in front of the vertebral column through a mesial gap. This gap when displayed is of such size that the tip of a little finger might be forced through it, but when undisturbed has little vertical height; and it is bounded by a continuous ring of cartilage, which above is in continuity with the occipital bone, and at the sides consists of the centres of the cervical and upper six dorsal vertebræ divided and crushed together; while inferiorly the remainder of the column is continued straight down, the laminae of the six lower dorsal vertebræ and the five lumbar being expanded out laterally. The scapulæ are crowded up into close contact with the skull, but all the five trunks of the brachial plexus emerge normally from the altered column.

*Cerebro-spinal axis*.—The disposition of parts, so far as examined in the region of the spinal cord, agreed with that found in other cases of open spina bifida, *e.g.*, I. and VI. A broad membranous area extends down the back, but the integument is complete over the sacrum. This membranous area is continued up beneath the tumour representing the brain, and the smooth surface is continued over that tumour as in cases of more complete anencephalus; but the membrane covering the tumour is tough, firm, and fibrous, though dark and smooth and unlike true skin. It exhibits a small perforation on its upper part. On raising the collapsed roof of the skull, there were first seen two hollow sacs, placed symmetrically, with little cerebral substance in their walls; and between and beneath these was found a larger and more thinly walled pouch projecting forwards mesially from

<sup>1</sup> Similar to that figured by Isidore Geoffroy St. Hilaire, *Histoire des Anomalies*, pl. viii. fig. 5.



the brain behind. The lateral pouches, probably olfactory bulbs, showed considerable dimensions when floated out in spirit, and the mesial pouch likewise was then seen to have a considerable cavity. Its membranous wall was so thin that it was much damaged in the removal, but the pituitary body adherent to it shows it to be the infundibulum. A delicate membrane also floats loosely in the angle beneath the tumour, in the position for the 4th ventricle. Thus there would appear to have been considerable effusion. Elongated fifth and other cranial nerves could be made out as in specimen III. Within the fibrous covering of the tumour, a very definite brain-structure is found. Two hemispheres project backwards and form its upper half, but, so far as exhibited, are solid; they are slender and become sharp-edged posteriorly (fig. 8). They overlie two lateral masses with smooth upper surfaces, which manifestly consist in greater part of optic thalami, though it might be difficult to decide how far corpora quadrigemina and other parts enter into their composition. Between these bodies a groove is continued down into the mesial hollow sac already described as lying on the base of the skull, so that the sac in question is manifestly an expanded infundibulum, like that of specimen III. Lifted up with the part of the brain raised by the section made, is seen a structure descending between the hemispheres, manifestly consisting of falx cerebri and other mesial structures. *Viscera.*—The adhesion of the dura mater in the neck to some viscus suggested the propriety of opening the visceral cavity (fig. 4). Beneath the liver in the abdomen are seen the two kidneys, and in the middle a slightly convoluted intestine continued up from the rectum, but neither stomach, spleen, small intestine, nor cæcum. From the umbilicus to the site beneath the liver where the intestines become invisible, extends, passing on the left side of the intestine, a long slender cord, one of the cords described by Allen<sup>1</sup> and recognised by him as vestiges of the omphalo-mesenteric vessels, remaining till after birth in various animals, but not found by him in the human subject at a period later than when the trunk from vertex to coccyx was 3 inches long. On laying open the thorax and neck, a tumour was found in the posterior mediastinum. It was cut into from the right pleura, and found

<sup>1</sup> *Jour. of Anat. and Phys.*, xvii. p. 59.

to contain a prolongation from the peritoneum ; and, covered by this, was laid bare the stomach which extended from the level of the lower bodies of the larynx down to the diaphragm, with the lesser curvature turned to the right and backwards. To the upper part of the lesser curvature is attached some tissue, which is that which has been adherent through the gap in the vertebral column to the dura mater, and appears to be connected with the pancreas. From the greater curve of the stomach, turned to the left, a peritoneal covering extended over a mass of intestines ; and having divided this, I find that it is the great omentum carried over the intestines so as to enclose them in a manner similar to that described by me as occurring in an anomalous instance many years ago.<sup>2</sup> The neck of the enclosing hood formed by the great omentum is, as in that instance, quite a narrow opening. It is situated at the pyloric end of the stomach, immediately above the opening between the crura of the diaphragm, by means of which the intestines have been drawn up into the thorax. The portal fissure is, as it were, pulled back to the posterior wall of the abdomen by the connection of the bile duct with the intestine just beyond the pylorus ; and consequently the lobulus Spigelii is drawn upwards so as to be visible from the thorax. The spleen is in its proper relation to the stomach, namely, at its cardiac end, which is situated in the neck. The cœliac axis and superior mesenteric artery are both given off in the thorax ; the inferior mesenteric and the renal arteries arise in the normal manner. The omphalo-mesenteric cord passes up into the thorax, and ends in the mesenteric vein.

V. *Encephalocele, Spina Bifida, and Anencephalic Skull.*—This is a female fœtus of six months. I need only mention the points which give it interest in connection with the other specimens. The back is much shortened, the whole of the dorsal as well as cervical vertebræ being involved in the part drawn up into a deep concavity below the skull. The membranous area over the open spina bifida extending to the base of the sacrum, has a quantity of vascular tissue in it, friable and difficult to dissect. Overhanging it is a large cerebral tumour, about the size of the rest of the head, and covered with tough smooth

<sup>1</sup> *Jour. of Anat. and Phys.*, May 1868.

covering like that described in specimen IV. On lifting this tumour by dividing the integuments in front of it, elongated fifth and other nerves were brought into view, and a mesial hollow structure, but no roof bones, the base of the skull being that of a typical anencephalus. On cutting open the tumour, it was found impossible distinctly to recognise any of the normal parts of the brain; but there was abundance of cerebral substance thrown into irregularities round a hollow prolonged forwards to the base of the skull, and attached in the sphenoidal region.

VI. *Anencephalus with Open Spina Bifida, and Small Cerebral Tumour.*—This is a female foetus of more than six months. It measures 7 inches from the upper end of the anencephalic membranous area of the head, down the back to the anus; and of this 5 inches are open cerebro-spinal axis, leaving 2 inches of sound skin between that and the anus. Half an inch above the anus is a post-anal dimple connected with the tip of the coccyx. On the fore half of the cephalic area is a hollow tumour the size of a cherry, with an opening a fifth of an inch in diameter at its back part. It is hollow, lined with a fine smooth surface thrown over a number of ridges and corrugations and processes of a soft substance evidently cerebral. The membranous covering of this tumour is thicker and whiter than that belonging to the spinal area. Behind the head the membranous area is bound firmly down by dense tissue, which has fitted into a depression behind the head, in which are involved some of the bodies of the upper dorsal vertebræ, while the cervical bodies are unrecognisable. The 1st and 2nd ribs are close up to the skull, and the angle of the scapula is crowded in between the skull and them. After dissecting off the integuments of the back along with the membranous cerebro-spinal area, the spinal ganglia of the dorsal and following nerves could be seen in two rows on the deep side of the parts removed; and, cutting down in the middle line between the spinal nerves, a continuous arachnoid sac was laid open in front of the anterior roots, which were seen arising near the middle line from the superficial membrane (fig. 9). Those, however, above the middle of the dorsal region could not be made out. The posterior roots of the spinal nerves come off in rows about a fifth of an inch from the middle line. But above the middle of the dorsal region these also are

absent, though the spinal ganglia can be made out. The arachnoid sac between the anterior roots is wide above; and, inserting the scissors external to the row of ganglia, I have on each side followed down a sac which corresponds with one-half of the part of the arachnoid space which normally lies behind the ligamenta denticulata. It is separated from the anterior sac, partly, as is usual, by the ligamentum denticulatum, and partly by an uninterrupted fold continued down over the roots of nerves. Superiorly, the arachnoid sac is traced up on each side of the dense tissue filling the hollow beneath the back of the head, and each half unites with its fellow on the base of the skull. Here also can be made out a basilar artery formed from two vertebrals and dividing into two posterior cerebrals, and, on each side, an origin of the fifth nerve; but other details I am unable to identify. Descending from the dense tissue at the infracephalic depression, is a pyramidal thickening of about an inch in length, whence proceeds downwards a mesial cord, which may be the anterior spinal artery, but it is not injected. On each side of this a finer line descends, which is probably the white band normally found in the ligamentum denticulatum.

VII. *Another Anencephalus with Open Spina Bifida.*—This specimen is a male about the same age as the last. Like it, it shows arachnoid sacs disposed between and beyond the roots of the spinal nerves. In this instance small lateral vascular masses are the only elevations on the base of the skull; but the arachnoid is traced forwards to the front of the cephalic part of the membranous area. The cervical vertebræ can be made out, although all fused together; and the cervical and upper dorsal nerves arise superficially as much as an inch below their emergence, that is to say, their origins from the membranous representative of the cord are situated towards the middle of the dorsal region, and in their course to their places of emergence they form a sort of cauda equina directed upwards.

VIII. *Anencephalus with Foramen Magnum completed.*—This is a fully developed female infant, with the neck and back normal, and a spongy mass occupying the membranous area over the head. On proceeding to remove this mass along with the skin round about from the skull, it was found that the occipital bone completed an arch over the foramen magnum, although

the two lateral parts which did so were yet unanchylosed in the middle line above. A mesial incision was made, and these two lateral parts prised asunder in order to remove the protected cranial contents along with the exposed spongy mass. The skull presents characters approaching those figured under the name of "podencephalus" by Geoffroy St. Hilaire in plate vi. of the 2nd volume of his *Philosophie Anatomique*, but differs from it, and much more from the podencephalus figured in plate ii., in having the occipital less complete and the base more exposed.

The spongy mass removed with the integument was found to be surrounded with a capsule of dura mater, and this being opened exhibited a falx well developed in front and coming to a point behind, while on each side, placed symmetrically, was brought into view an elongated vesicle stretching by the side of the falx, and another extending outwards from its fore part. These vesicles were embedded in dense connective tissue laminated and vascular, an obvious development of pia mater, and on being opened were found to be entirely devoid of nervous substance. From beneath the protection of the occipital arch a perfect medulla oblongata has been removed, but it ends abruptly above, not far from the posterior extremities of the longitudinal vesicles; and there is no vestige of cerebellum, the sides of the posterior fossa basis cranii having been occupied by two masses of tough leathery substance instead. The cervical vertebræ and the muscles at the back of the neck are normal.

IX. *Anencephalus without Supra-Occipital, and without apparent Spina Bifida*.—Fully developed female infant. In this instance there seemed to be scarcely any spongy mass beneath the membranous area. The little that was detected was in front, but the petrous bones seemed covered with mere membrane. Behind this, near the back part of the membranous area, which was confined to the head, a pit was detected which proved to be a foramen passing down into the interior of the cord. The spine of the axis was normal, with muscles normally attached; but the posterior arch of the atlas was incomplete, a state of matters not very uncommon in otherwise normal subjects. A few of the cervical spines having been split up, the upper part of the cord was removed along with the membranous area which was stripped from the skull. The slight intumescence of this area

in front was then found to depend on a quantity of tough blood-stained tissue which adhered to the fore part of the exposed base of the skull. The clivus was found, when traced downwards, to be bent forwards under the skull, completing, with the bodies of the vertebræ beneath, a recess in which the medulla oblongata was lodged, and lying at an angle less than a right angle with the upper surface of the body of the sphenoid, which also, as is habitual in anencephalus, projected upwards between the petrous bones, by reason of the outer ends of these latter being depressed. On splitting the dura mater in front of the cord upwards, the arachnoid space in front of the nerve-roots was laid open and the medulla oblongata exposed, crossed above by a delicate transverse band representing the pons Varolii, while on each side was a delicate structure readily recognised as the flocculus, but not a vestige of cerebellum.<sup>1</sup> The arachnoid sac was pursued further forwards, spreading out to each side in a position corresponding with the prominent edge of the petrous bone. Towards the fore part of the membranous area a small opening in the middle line was found overhung by spongy substance, and leading into a sac about an inch and a half in breadth and half an inch in length, with two smooth symmetrical masses, united by a narrow isthmus, projecting from the roof, obviously representatives of portions of the brain, doubtless the hemisphere vesicles. Thus in this specimen there were two openings from the membranous area, one leading back into the interior of the medulla oblongata, and the other forwards to the termination of the cerebro-spinal tube in front.

In addition to these nine specimens, selected from among a number of human malformations presented to my predecessor and myself, I shall describe two specimens of chicks, the importance of which might have escaped my notice had it not been for the instructive memoir published in 1881 by Dr. Lebedeff,<sup>2</sup> in which he describes various specimens of anomalous brain and

<sup>1</sup> The flocculus, though always described with the cerebellum, is very well understood, by those who have studied its development, to be totally unconnected with that organ, being developed from a lateral outgrowth of the floor of the 3rd cerebral vesicle, while the cerebellum is developed further forwards from the foremost part of the roof of that vesicle.

<sup>2</sup> "Ueber die Entstehung der Anencephalie und Spina bifida bei Vögeln und Menschen," Virchow's *Archiv*, lxxvi. p. 263.

spinal cord in the embryo chick, and points out their bearing on anencephalus in the human subject.

X. *Embryo Chick* (fig 10).—This specimen exhibits the open condition of the cerebro-spinal canal found in the first day of hatching. But the open condition is in this instance anomalous, for there is an overgrowth of the cerebro-spinal axis; the parts apparently corresponding with the greater part of the cerebral vesicles being much thickened, and the part immediately behind, namely, that which appears to represent the anterior cervical region, being thrown into numerous plications as well as expanded, while the expanded condition is carried back the whole length of the embryo.

XI. *Embryo Chick; Cyclopia* (figs. 11 and 12).—This is a chick hatched for nine or ten days. The principal peculiarities are confined to the head, which is turned to the right side and surmounted by exposed brain forming a lobulated mass overflowing the base of the skull, and consisting of an open ventricle with a largely developed laminated mass on each side, apparently the right and left moieties of the cerebellum, and smaller masses in front of these. On the surface there was no appearance of eye nor of ear, and instead of a beak there was an elongated projection with an irregular aperture at the extremity, which suggested the idea that it was such an orifice as is seen in agnathia of mammals, combining mouth and ears in one. This view was corroborated by opening the orifice up, when one side of the lower jaw was found ending in a point at the side of the aperture. Also, in the floor of the cavity, when cut up, was seen a pimple representing the tongue. But what was most remarkable was a minute cyclopic eye, detected by being black with choroidal pigment, but minute, and situated so deeply that it could not be seen till the dissection was made.

#### GENERAL REMARKS.

By placing together more or less detailed accounts of the anatomy of nine different instances of imperfection of brain and spinal cord in the human subject, there is exhibited the close connection between conditions conveniently described by different names, and even yet too often looked on as thoroughly distinct.

The first impression, on looking at numbers of monstrosities, is that the different groups are each one remarkably distinct from others, while its members are remarkably similar. It is afterwards that the links between them strike us; and one quite understands the reasons which led the great Geoffroy St. Hilaire to believe them capable of being distinguished in a way perfectly similar to that in which zoological genera and species are determined. It is not to be forgotten that his work in respect of monsters was governed by a desire to develop a philosophy of anatomy. He saw in monsters, as in zoological genera and species, the operation of external surroundings on the internal formative impulse of the organism, or, so to say, of environment on heredity; and if he had not the opportunity of appreciating the connecting links between diverse forms of monstrosity, he was the first to suggest the possibility of change being effected by environment in zoological species, to which he considered that monsters stood in an appreciable relation. It may further be noted here that the considerations about to be laid before the reader in the remainder of this memoir point out that the principle put forward by St. Hilaire, namely, the direct action of environment on form, is the source of modification of the embryo by sudden change in individual instances to such an extraordinary extent as to suggest that its influence by gradual and continued change operating on the embryo in successive generations cannot be easily estimated. The principles enunciated by Lamarck, St. Hilaire, and Darwin respectively, namely, the hereditary transmission of the effects of habitual actions, of environment, and of natural selection, all have their places in nature. As to whether any of them or all combined are sufficient to produce higher forms from lower that is a different question, and one not likely to be settled by dogmatic asseveration or even the insulting jibes which occasionally disgrace the London press.

Where St. Hilaire went wrong with regard to monsters seems to have been principally in making an arbitrary distinction between pathological action and arrest of development, and denying the pathological element; apparently not sufficiently appreciating that pathological and physiological action are both excited by stimulation from without, and only differ in respect that in pathological action the normal sequence of events in the



organism is altered. An arrest of development presupposes some cause of the arrest, and, according as the cause varies in character, time, site, and degree, so do its effects vary, without being so specifically distinct as they at first appear.

In the first of the specimens now described an open spina bifida, or complete "rachischisis" of the lumbar region, is seen in association not merely with hydrocephalus, but also with a dilated condition of the central canal in the intervening cord. In the second specimen a cervical spina bifida covered with natural integuments presents want of closure of the central canal and of the vertebral arches, and also shortening and bending forwards of both cord and column, accompanied with a certain lateral expansion. In the third specimen, while the brain is displaced and the skull deformed in consequence, the changes in the cervical part of the column are of a similar character to those in the previous specimen, while there are again unmistakable signs of accumulation of fluid, though in this instance intracranial. In the fourth specimen open spina bifida in the whole length of the cord accompanies a displacement of the brain very similar to that in the third specimen, though uncovered with true skin, and with the occipital bone less developed. In the fifth specimen an open spina bifida is surmounted by a cerebral tumour similar in appearance to that of the previous specimen; but when the tumour was opened into, the brain was found to be less developed; and the skull was that of a typical anencephalus. In the sixth specimen we have an anencephalus with a small cerebral tumour perforated near the back, so as to be continuous with the open spina bifida; and in the seventh specimen complete anencephalus with open spina bifida is illustrated in simplicity.

The eighth and ninth specimens are instances in which the lesion is confined to the skull and its contents; and it is noticeable that while in both there is a medulla oblongata, the brain is a shade less interfered with in specimen nine, in which the skull is completely unroofed and enormously bent out of shape, than in specimen eight, which has occipital elements roofing over the medulla oblongata. This is an additional illustration that interference with the brain and interference with the skull do not march *pari passu*.

## ORIGIN OF ANENCEPHALUS AND SPINA BIFIDA.

The connection of these anomalies with dropsies in the embryo has been maintained since Morgagni wrote till now. Geoffroy St. Hilaire refers to Meckel and to Tiedemann as recognising in such affections an arrest of cerebral development, and has elaborated a like view, holding that there is an accumulation of fluid, but that this is not the result of disease but the persistence of the embryonic condition when fluid is normally contained within walls in which the nervous substance is not yet developed. It is plain, however, that St. Hilaire believed in the existence of a membranous pouch containing between two and three litres, or about half a gallon of water, attached round the margins of the membranous area of the specimen of anencephalus and open spina bifida which he was describing, although he saw no vestige of such a thing himself, but managed to get an account of it, vague enough, from the midwife, after having arrived at the conclusion that such a thing must be.<sup>1</sup> That such a pouch actually did exist in that particular instance I am not prepared to deny, seeing that it is perfectly conceivable, and would furnish an intermediate condition between spina bifida completely open and that which is covered with true integument, but undoubtedly in none of the many instances of spina bifida and anencephalus that I have seen have I detected traces of such a thing, and in none that are now within my observation is there the slightest interruption of continuity between the integument and what I term the membranous area. Isodore Geoffroy St. Hilaire adheres to the views of his father, and speaks of the integuments being replaced before birth by a "vast hydrorachic tumour," of which nothing is left but irregular débris.<sup>2</sup>

The views of Dareste appear to coincide entirely with those of St. Hilaire. He speaks of the brain and spinal cord being replaced by a pouch full of serosity, at some distance from which the integuments cease.<sup>3</sup> He calls it a physiological dropsy, and says "this accumulation of serosity hinders the

<sup>1</sup> *Philosophie Anatomique*, ii. 131.

<sup>2</sup> *Histoire des Anomalies*, ii. 360.

<sup>3</sup> *Production Artificielle des Monstruosités*, 1877, p. 248.

formation of the nervous substance." He further suggests, as the initial deviation from normal growth in anencephalus, pseudencephalus, and exencephalus, an arrest in the development of the "cephalic hood which causes it to remain applied to the superior aspect of the head, and to compress it in part or altogether,<sup>1</sup> and in this idea he is followed by Perls<sup>2</sup> and Marchand.<sup>3</sup>

Förster describes anencephalus and spina bifida, or, as he terms them, cranioschisis and rachischisis, under the head of *Hydrops canalis medullaris*, and describes them thus:—"The original collection of water is so considerable that a fissure is thereby effected of the osseous walls and coverings of the medullary canal, and a more or less perfect destruction of the brain and spinal cord." He states also that the dura mater and other membranes of the brain in anencephalus form a great sac which covers the whole base of the skull, or bursts so as to leave the base covered with the exposed remnants of the membranes and of the brain.<sup>4</sup>

Ahlfeld,<sup>5</sup> while admitting that other causes combine to produce anencephalus, considers that none can compare in frequency with cerebral dropsy; and he dismisses as unsupported the views brought forward by Lebedeff. Lebedeff's memoir, however, is one of the utmost importance. For it will be seen that in all previous views an accumulation of water has played an important part, whether looked on as a purely pathological accumulation or not; but Lebedeff points out among other things, and figures abnormal conditions of the young embryo chick, in which closure of the cerebro-spinal cylinder has failed to take place. The blastema for the roof of the skull and spines of the vertebræ fails in these cases ever to reach the middle line; and there can be no thought of their having been turned aside by the pressure of a bag of fluid. Lebedeff figures three such cases, and gives vertical sections of others, showing that there is an undue thickness of the medullary plate, and that complicated foldings of it occur, with consequent formation of adventitious vesicles. He concludes that anencephalus occurs in consequence of changes of

<sup>1</sup> *Op. cit.*, p. 254.

<sup>2</sup> *Lehrbuch des Allgemeinen Pathologischen Anatomie*, p. 283.

<sup>3</sup> Referred to by Ahlfeld, *op. cit.*, p. 290.

<sup>4</sup> *Op. cit.*, pp. 77 and 80.

<sup>5</sup> *Op. cit.*, p. 290.

the medullary plate in the earliest stages of embryonic life, either while as yet the medullary furrow is open or at a later time; the tube either having never been closed or having flattened out, and its hinder wall given way.<sup>1</sup>

Importance is due to the observations of Lebedeff, which are supplemented by Specimen X. of this communication, first, because they demonstrate that failure in closure of the cerebro-spinal axis is a thing which does occur, and therefore which may be found in an older embryo as well as a younger; and, secondly, because the development of nerves in regular series, and of both anterior and posterior divisions of the arachnoid sac, is not likely to have gone on with uninterrupted regularity under the pressure of a collection of fluid so destructive in its effects as to have destroyed by pressure the brain or spinal cord. The regular origin of the nerves was brought forward by Geoffroy St. Hilaire as evidence against a pathological collection of fluid; but, so far as I know, the circumstance of the portion of the arachnoid sac normally posterior to the roots of nerves being developed in two divisions external to them has not ere now attracted notice. Its significance consists in this, that while the substance of the spinal cord is represented by no more than an epithelium, the part of the arachnoid which should have been behind the cord pursues its development in its displaced position exactly as is the case with the laminæ of the vertebræ.

While, however, anencephalus and spina bifida exhibit deficient development of brain and spinal cord, the chicks described by Lebedeff present over-development of the medullary blastema; and in Specimen X. above described, such over-development is shown in two ways, namely, by thickening and by plication. But the medullary blastema would not be plicated were it at liberty to extend at an increased rate longitudinally. An equally increased growth of all the parts would simply yield a larger embryo of normal form. The plications arise from connection with a mesoblast growing at a slower rate. So also, in one of the embryos figured by Lebedeff, though there is no plication, the undue elongation of the fore part of the medullary plate appears to have produced increased cephalic curvature or kyphosis, and the same thing has happened in the embryo

<sup>1</sup> *Op. cit.*, p. 297.

chick, Specimen XI., above described. Lebedeff, indeed, points out that flattening of the brain part of the medullary plate would be a mechanical consequence of increased curve, just as one flattens a half tube of india-rubber by bending it backwards; and this may be considered as an element having its value; but it is not necessary to resort to any other explanation than the overgrowth to account for patency of the tube. In another somewhat older embryo figured by Lebedeff, one in which the limbs have appeared, the whole embryo is thrown into a letter S curve in a manner common enough in artificial incubation, apparently because the surrounding membranes have cramped it longitudinally. But the undue expansion of the medullary plate is not only longitudinal; it is likewise transverse; and if this transverse expansion be especially in the superficial layers, it will manifestly exercise an everting influence on the sides, and prevent their turning back in the normal way to meet in the middle line. The normal inversion of the edges of the plate is produced by a comparatively greater growth of the deep than of the superficial strata, and an equal growth of both would be sufficient to keep the furrow wide open.

Recalling to mind what a fruitful source of abnormalities unequal temperature is in artificial incubation, a subject to which both Dareste and Panum have given special attention, we need have little hesitation in referring numbers of abnormalities to the effects of accidental stimulus acting on the whole embryo or on a part; and if it further be kept in mind, that while gentle and recurrent stimuli may increase the vital energies, a sudden and prolonged exaltation of action is followed by exhaustion, we need not, I think, deem that there is necessarily any improbability that abnormalities presenting deficient development of parts have originated in the excessive growth of those parts at a very early period.

The abnormalities under consideration in the chick have all the appearance of depending on undue stimulation, at a very early date, of the whole or part of the cerebro-spinal axis; and the effects so produced we now see may be rationally considered sufficient to prevent the closure of the neural cylinder. We may go further, and *even if we shall find reason to believe that spina bifida and anencephalus result from the rupture of an*

*already closed cylinder, refer the abnormal condition to overstimulation in the first instance.* The pouring out of fluid by living corpuscles is an effect produced by stimulation carried on in some particular manner and to a certain degree, though of the *modus operandi* in detail we know little or nothing. This is illustrated by the action of vesicants. The collection of fluid in an already closed cerebro-spinal cylinder is doubtless also an effect of over-stimulation of the living corpuscles in a certain manner and degree. The proliferation of these corpuscles in a manner such as to produce tissue of healthy consistence, but of undue amount, is likewise the result of external irritation or stimulation acting on the internal impulse or vital properties; but the adjustment of the two factors is in different proportion. Thus there need be no radical distinction, so far as the first cause is concerned, between an abnormality produced by non-closure of the medullary tube, and one due to a collection of fluid at a slightly later period. In both instances there is undue action in the originally superficial tissues of brain and spinal cord. In the one case the increased growth everts parts which ought to turn inwards, in the other the collected fluid pushes back the edges which had already become united.

Recognising, therefore, that abnormality may originate in both of these ways, seeing that on the one hand non-closure of the cerebro-spinal tube actually occurs in embryo chicks, while on the other spina bifida with complete closure of the integuments in the middle line is common, the question to solve is, whether in anencephalus and open spina bifida the abnormal conditions have originated before or after the period for closure; and this can only be determined by attention to the evidence afforded by the details of their structure.

From this point of view Specimen I. is highly instructive. Here you meet with actual hydrocephalus combined with an open lumbar spina bifida, the margins of which exhibit no trace of a rupture, but an uninterrupted passage from the normal epidermis to the delicate covering of the membranous area, and the arachnoid arrangements opposite the spina bifida are completely developed without trace of any result of inflammation. This is exactly such a spina bifida as might be supposed to originate from non-closure. But we have seen that, continuous

with its upper part, there is a dilated central canal where the spines and spinal cord are complete, and even the peripheral appearance of the cord remained normal, notwithstanding the dilated canal. This dilatation of the canal may indeed have arisen from want of normal development of the nerve substance surrounding it, but as one might expect that such a want of development would have arrested the growth of the cord in diameter as well as in the thickness of its proper substance, it is perhaps more likely that distension of the canal, after closure, caused its dilatation above, and led to its rupture below in very early development, while as yet there was little texture deposited in the middle line. The hydrocephalus was obviously of much later origin, when the different parts of the brain were already formed.

But more important than the teachings of this single specimen is what is to be learned from the consideration that in anencephali generally the eyes and their surroundings are normally developed. The primary optic vesicles are not separated from the vesicles of the brain till considerably after these latter have been roofed in ; and the evidence already given that structures are modified in their development by alterations in their mechanical relations, together with further evidence to the same effect about to be given, makes it very improbable that hollow organs ordinarily developed in connection with a closed cavity would exhibit the normal form in instances in which that cavity remained open.

The eyes of anencephali have been made the subject of special investigation by De Wahl and Manz,<sup>1</sup> and their only deviation from normal development consists in the absence of nerve fibres from the optic nerve and retina. That, no doubt, is a circumstance in itself very interesting ; it contrasts with the full development of the spinal nerves, and may suggest a question as to whether embryologists are right in translating the beautiful observations of F. M. Balfour on the early connection of the spinal nerves with the cord, as sufficient proof that these nerves are epiblastic in origin. But, taken in connection with the otherwise normal development of the eye, it cannot be looked on as showing more than that the rupture of the cranium in

<sup>1</sup> Manz, Virchow's *Archiv*, li. p. 315, where also reference is made to E. de Wahl.

anencephalus has occurred before the development of nerve fibres in the optic nerves and tracts. I apprehend, therefore, that *anencephalus most probably results from rupture of closed parts after the optic nerves have ceased to be tubes of communication between brain and eye*, and that such a form as the chick, X., the result of stimulation at a considerably earlier date, would be doomed to perish in the early stages of development. Panum<sup>1</sup> figures a chick hatched for 112½ hours, but of a development which suggests not more than sixty hours, with a beautiful broadly open lumbar spina bifida. I am not prepared to say that such a case as that has not been produced by non-closure, but lumbar spina bifida differs in some very important points from anencephalus and cervical spina bifida.

Lumbar spina bifida is not usually associated (I suppose, indeed, never is) with shortening of the vertebral column at the part, while there would appear to be always shortening of the column with cervical spina bifida; and anencephalus when complete is associated with cervical spina bifida. On the hypothesis, then, of undue growth, in the early embryo, of the medullary plate as compared with the subjacent structure, we shall see in lumbar open spina bifida the result of thickening or transverse growth, but without evidence of abnormal elongation; while in reference to cervical spina bifida the question arises, how far the elongation may be merely comparative, and arrest in elongation of the notochord be the real abnormality. As far as I can see, *the phenomena of cervical spina bifida can be accounted for by mere want of extension of the notochord*, while the medullary plate grows on at the normal rate; but since it is certain that very early overgrowth of the medullary plate does occur while as yet the parts for the dorsal and lumbar regions are but little developed, it appears very probable that the same stimulus which has that effect is also the cause of the non-elongation of the notochord. For this failure of the notochord to elongate is not to be accounted for by mere absence of its structure, which would offer no resistance to the elongation of the parts around but must be the result of an abnormal deposition firm enough to bind the surrounding parts to some extent.

<sup>1</sup> "Untersuchungen über die Entstehungen der Missbildungen, Zunächst in den Eiern der Vögel," 1860, Taf. vi. fig. 8.



Suppose the notochord to refuse to lengthen while the centres of growth for the bodies of the vertebræ have still the normal tendency to increase in size, the elongation of these will bend them outwards, making it more difficult for them to unite in the mesial plane, and thus broadening the bodies of the vertebræ, or pushing their right and left parts asunder, as we have seen occurs.

But what, then, is the cause of the convexity forwards of the shortened part of the column which is uniformly found accompanying cervical spina bifida? It is explained at once by the visceral arches being normally developed, though crowded on a shortened base. The effect of a comparatively large growth on the ventral aspect of the arrested notochord will be to bend the embryo back, on the same principle that growth on the dorsal aspect at an earlier date bent it forwards. But when the shortening of the column is very considerable, the bending backwards is insufficient to give room for the normal amount of growth of the dorsal and ventral plates, and I suppose they are thus constrained to continue further in a ventral course, leading to the length of ribs and great antero-posterior diameter of the thorax which are found in such cases. Another mechanical factor may, however, co-operate to that result. The rotation outwards of the walls of the neural canal involves the rotation forwards and inwards of the outer border of the dorsal plates, and a consequent diminution of the transverse diameter of the thorax, the ribs passing more directly forwards. One would anticipate that the same inward rotation of the visceral and maxillary lobes of the neck and face would favour the union of these parts in the middle line at a somewhat earlier date than usual, and lead to a certain upward pressure of the parts within their circle. On the other hand, the vault of the skull having been ruptured, there is an absence of that pressure downwards which is exercised in normal circumstances by the growing brain pressing on all its surroundings. It is owing to these causes that in anencephalus the base of the skull is bent downwards at the sides, as has been correctly noted by various authors. Perls alludes to this circumstance as an argument against dropsy as the cause of anencephalus; and it is a valid argument against a later dropsy being the cause, but not against an expansion leading to very early rupture of the vault.

On the hypothesis that all the abnormalities which we have been considering are consequences of over-stimulation in early embryonic life, it is obvious from what has been said that much difference of result will depend on the exact period at which the stimulus is applied. Speculating in a more tentative manner, I incline to believe that such over-stimulation, occurring soon after impregnation of the ovum leads to fissiparous division of the germinal mass, resulting in two embryos; that at a later period, or carried to a slighter extent, it will cause partial division, resulting in such forms of double monster as have a portion of the cerebro-spinal axis undivided; that at a later period, when the cephalic end of the neural furrow is formed but is still open, it produces such conditions as we have seen in the chick X., but that, in consequence of interference with their whole form, embryos so affected probably always perish early; that anencephalus is produced by rupture after the primary optic vesicles are cut off from the cerebral vesicles; and that lumbar spina bifida occurs later.

In an attempt to account for so many things by similar causes, one naturally asks, What is the relation of cyclopia and pericephalus to anencephalus? On that subject we may get some light when we study the conditions of the brain, as we must now do, to complete our view of the causation of the latter.

#### THE BRAIN AND CORD IN ANENCEPHALUS, ENCEPHALOCELE, AND SPINA BIFIDA.

I have already drawn attention to the very general presence of the arachnoid sac, and have thereby removed all possible ground for doubt that the membrane stretched instead of skin over the base of the skull in anencephalus, and over the vertebræ in open spina bifida, is in the main the surface of the medullary groove which has either remained open from the very first, or, as I think is more frequently the case, has become reopened by redundant collection of fluid and growth of substance very soon after closure. Thus, it is easy to recognise that a membranous area on the back is an enlarged central canal of the spinal cord, and that the texture between the surface and the arachnoid sac, however thin, contains whatever represents the substance of

the cord and the pia mater; and, indeed, a certain development of pia mater may render it spongy and vascular. The masses of spongy vascular tissue surmounting the base of the skull in most cases of anencephalus are nothing else than redundant pia mater. But we cannot consider the membranous surface on the skull of an anencephalus as corresponding in all cases, or even in most, altogether to the interior of the ventricles of the brain, seeing that in many instances a hollow vestigial brain is present, and the hollow in these instances is the true ventricular cavity of the brain. Nevertheless, such a vestigial brain, however much it may approach full development, is never covered with true skin, but only with dura mater, and a transparent membrane adherent thereto. That transparent membrane, therefore, must be looked on as a sort of adventitious outgrowth of the lining of the central canal and ventricles of the brain, beneath which the dura mater is continued round towards the middle line. Specimen VI. illustrates very well such a state of matters.

The very existence, however, of a hollow brain indicates a state of matters not touched on in the preceding remarks on the causation of anencephalus. It indicates that while the roof of the skull has been ruptured, there has been an unruptured part of the roof of the brain. In Specimen VI. the foramen at the back of the hollow tumour indicates where the wall of the brain has given way. This must have happened before the rupture of the surrounding parietes from which the integuments and bones take rise, for the regular development of the arachnoid space renders it unlikely that there had been effusion in the embryo between the cerebro-spinal axis and the parietes, and the gratuitous supposition that such a thing had occurred would in no way remove the necessity for supposing that effusion within the hollow of the axis had existed as well. It would appear, therefore, that a small rupture of the brain wall has relieved the pressure in the ventricles, while the superficial parts, continuing to be pressed on, and being much thinner along the middle line than elsewhere, are soon afterwards ripped up the middle more extensively. Probably this is the sequence of events in every instance in which the brain is represented by a vesicle with corrugated inner wall, as in Specimens V. and VI. and also IX., for the corrugations seem to indicate relief from internal pressure.

But every brain uncovered with true skin has not necessarily undergone rupture; it may by distension and displacement have ruptured the integument, while remaining itself unruptured. It seems probable that this is what has occurred in Specimen IV.; and, in fact, that case has much resemblance to the occipital encephalocle, Specimen III., save that it is complicated by open spina bifida, which very likely aided the rupture of the integuments of the head by giving a notch from which the tear may have extended under the pressure of the brain.

What, then, are the peculiarities of occipital encephalocle as illustrated by these two cases? They agree in the exaggerated development of the infundibulum, and the dropsical pouch on each side in front of the hemispheres; and in these points may be found sufficient explanation of their whole cephalic structure. The lateral pouches appear to be dropsical olfactory bulbs. The infundibulum is a prolongation of the thalamencephalon, from which the olfactory bulbs likewise take origin. There has been dropsy, therefore, of the thalamencephalon before the closure of the communication with the olfactory bulbs; and this dropsy is seen in Specimen IV. to have taken place after the walls of the second cerebral vesicle were sufficiently strong to resist its distending force, and to have pressed on the hemispheres so as to interfere with their growth, while in Specimen II., no doubt, it had entered the hemisphere vesicles and distended them. The hinder part had still been the portion of the roof of the skull least securely united in the middle line, and thus gave way before the pressure with which the brain was pushed back by the dropsical distensions in front, which were thus left in possession of the cranial cavity.

That dropsical enlargement of the olfactory bulbs and infundibulum should thus give rise to occipital encephalocle is exceedingly remarkable when taken in conjunction with what I demonstrated in a former memoir in this *Journal*,—that in cyclopia there is dropsical enlargement of the roof of the thalamencephalon, including the pineal body.<sup>1</sup> But in cyclopia there is not only a mesial eye, but also a single vesicle or mass to represent the hemisphere-vesicles; also the infundibulum, so far from being enlarged, is sometimes absent, and there is no

<sup>1</sup> "On the Brain of Cyclopia," *Jour. Anat. and Phys.*, xii. 518.

vestige of olfactory bulbs. Further, the dropsical vesicle found in cyclopia is also found in agnathia or perocephalus, but in that form there are the further peculiarities that the nose and eye have disappeared altogether, as has also the mass representing the hemisphere-vesicles, and that the first visceral arch, as well as the middle and lateral frontal processes of the embryo have been imperfectly developed, and that the clefts in connection with the external ears remaining open, and being approached one to the other, are undifferentiated from the buccal orifice. One other set of facts we must take into account, namely, that in cyclopia and perocephalus the vault of the skull is complete, and the hinder parts of the cerebrum normal, as are also the spinal cord and its coverings. To attempt to explain these things is to launch still further into the regions of hypothesis than I have been already obliged to do; still I think that the road to a rational explanation may be pointed out.

The unaltered condition of the corpora quadrigemina and parts behind it points to these parts being 'shut off sufficiently from the parts in front to preserve their form. As the infundibulum is not enlarged, while the region of the pineal body is certainly the main seat of the distension, the constriction must be between the two, a position which exactly corresponds with the optic tracts.<sup>1</sup> The cyclopic dropsy is probably of later origin than the anencephalic, and cannot on that account split up the parietes enclosing the brain behind. On that very account it does more injury in front, and induces a kyphosis or downward curvature, as well as a rotation inwards of the lateral parts for the growth of the face. Nor can these effects be counteracted as in anencephalus by bending backwards of the neck, seeing that the cervical part of the column is closed in and resistant. Thus in cyclopia the primary optic vesicles, which, according to this theory, must have been already formed, are crowded together, to be surrounded by a common globe, and the frontal processes pushed forwards as a proboscis in front of the mesial eye so formed; while in perocephalus the crowding of parts

<sup>1</sup> Personally I have little doubt that embryologists are mistaken in considering the infundibulum as belonging to the thalamencephalon. It probably is really mesencephalic in origin. For in the embryo chick of about thirty hours one can see that the optic commissure corresponds in position with the constriction between the first and second cerebral vesicles.

having been greater, the suppression of structures is carried to a greater extent.

The chick, Specimen XI., is of special interest because, unlike cyclopia in mammals, it combines a ruptured brain and cranium with a cycloplan eye, and the fusion of buccal orifice and external ears with the presence of a lower jaw.<sup>1</sup>

In bringing these remarks on the cerebro-spinal axis to a close, I may note that there is still considerable difficulty in accounting for the total absence of the cord in open spina bifida. Four causes might be mentioned as possible agents, namely, exposure, pressure, exhaustion of the tissue elements, and stretching. Mere exposure I cannot entertain as a cause which could result in the smooth and entire membrane which we find in place of the cord, for the liquor amnii could scarcely be more detrimental to the growth of nervous tissue than the fluid in such a cervical spina bifida as Specimen II. Nor can pressure account for the phenomena, as the pressure, where there has been any, is removed at an early date. The destructive effects of stretching I have in a previous memoir shown in the case of appended monsters.<sup>2</sup> But stretching by the growth of neighbouring parts means comparative arrest of growth in the part itself, and thus we are thrown back on the fourth alternative, that such arrest really takes place in consequence of the proliferating power of the texture having been previously exhausted by overaction. Still the explanation seems scarcely sufficient, unless we suppose that the arrest in growth, and stretching of structure so arrested, prevent the development of blood-vessels.

#### STRUCTURES ENLARGED IN THE SPECIMENS DESCRIBED.

A most obvious instance of enlargement of structure is seen in the elongation of the cranial nerves in the cases of encephalocele. Here we have an example of nutrition keeping pace with stretching, beautifully seen in Specimen III., and contrast-

<sup>1</sup> As the hatching of an anencephalic chick has been considered as a rarity, I may note that I have been presented by Mr. John M. Campbell with a chick fully hatched which is double bodied, and with one neck and one anencephalic head.

<sup>2</sup> "On Double-Bodied Monsters, &c." *Jour. Anat. and Phys.*, viii. 255.

ing with the destruction of texture by the same cause in the upper part of the medulla oblongata of the same specimen. The same sort of elongation of nerve trunks occurs in the upper spinal nerves of Specimen VII., and is not uncommon. In this instance, also, the elongation arises from stretching of the nerves between their origin and their exit, and seems to indicate that in later development the superficial membrane in the lower part of its extent had refused to elongate farther, and had thus stretched the upper nerves.

The most extraordinary instance of enlarged structure is, however, the enormous laminated tubercle, three quarters of an inch long, of the cerebellum of Specimen I. Here is a structure in ordinary circumstances closely pressed on by other parts, which in this instance has obviously hung in a larger space caused by effusion of fluid. The fluid has pressed upwards on the cerebellum, and interfered with the normal development by means of which the edge of the posterior velum, originally directed downwards, comes to be turned forwards. But the laminated tubercle hanging free in this fluid has been very differently situated from what it usually is when its growth is resisted by solid structures. The mere absence of accustomed resistance has been shown by my friend and senior demonstrator, Dr. Allen, to be sufficient to lead to elongation of bone in the case of the head of the radius and the odontoid process;<sup>1</sup> and in this instance there has been removal of resistance of solids preventing elongation, and the pressure of fluid has been substituted, pressing equally on equal surfaces and in a direction at right angles to those surfaces.

Another instance of enlarged structure in the specimens described is found in the expansion of the vertebral laminae in open spina bifida in the dorsal and lumbar regions. The phenomenon is not met with in the cervical region, because the vertebrae there are crushed by the bending which takes place. But the constancy of the shape of the large flat plates into which the dorsal and lumbar laminae are developed is very remarkable. It is such a constancy as is comparable with the effects of heredity,

<sup>1</sup> "On Anatomical Changes induced by Dislocation Backwards of the Head of the Radius in Early Life," *Glasgow Med. Jour.*, July 1880; and "On Tertiary Occipital Condyle," *Jour. Anat. and Phys.*, xv. 65, Oct. 1880.

and recalls to mind other instances of a given interference altering in an explicit manner structures from their hereditary form. Of this sort are the effects of gall insects on different plants; also the growth of calcified corda dorsalis in renewed tails of lizards; phenomena which ought to stimulate the reflection of any one who may be disposed to explain every extraordinary structure which he meets with by deriving it from a purely hypothetical ancestry. Truly, we shall not find the full virtue of the law that environment modifies living forms, unless we appreciate, as has never been done, that it applies to every textural unit of the body, and that every vital action is the result of living particles and their surroundings acting and reacting one on the other. In this instance, the circumstance that the blastema from which the vertebral laminæ have been early derived has been thrown outwards must have modified the pressure to which they have been subjected, especially on the surfaces usually internal, but by eversion becoming superficial.

Another instance of enlarged development as a collateral consequence of a larger deficiency is seen in the enlargement of the mesial palatine processes of the intermaxillaries in connection with the cleft palate of Specimen II. They have beautifully expanded to form together a broad groove on which the vomer rests, exactly as one sees in the carnivora (fig. 3). This is the typical form of this part in mammals, but it is not found in normal circumstances in man, neither is it found in apes nor in monkeys. Yet, remaining potentially present, and as if suppressed for so long by mere mechanical causes, the peculiarities of this morphologically important part of the skeleton spring into view when these restraints are accidentally removed.

In this place I may also point attention to a phenomenon to some extent illustrating the converse of the principle with regard to pressure of which I have been furnishing examples. I allude to the collapsed cranial vault in anencephalus. When rupture has been complete, the blastema from which the roof bones, such as the parietals, is to be developed is quite displaced, and one does not wonder that when the ossification appears it should take the form of a thicker and unexpanded bone; but in cases such as Specimens III. and IV., where there has been no rupture in the frontal and parietal regions, one sees the effect of



the absence of the normal distending influence on the vault exercised by the growth of the brain. So also in Specimen II. the vault is small from want of growth of the brain, while in hydrocephalus the normal centres of ossification form more expanded plates than usual, and new centres of ossification arise around them to supplement them when they fail.

#### THE DISPLACEMENT OF VISCERA IN SPECIMEN IV.

Thoracic hernia of abdominal viscera is by no means unknown. Indeed, it occurred in Specimen II., but, owing to an accident, the parts were destroyed without being properly investigated. In an interesting account of a remarkable case of hernia diaphragmatica spuria sinistra, Wenzel Gruber<sup>1</sup> has given references to a number of cases of that sort previously described. But in the affection so named, the œsophagus passes into the abdomen, while the intestines enter the thorax by a deficiency in the left side of the diaphragm; while in the specimen which we have before us, although there is in like manner a large part of the alimentary canal lodged in the thorax, the cause and the details are totally different.

The commencement of the stomach in the neck at the level of the larynx is to be explained by recollecting that in very early foetal life the stomach is seen lying longitudinally in the visceral cavity, with its œsophageal extremity immediately below the bifurcating pouch which is the first indication of the respiratory organs. The lungs, as they develop, descend into the visceral cavity, and the heart descends with them, while the stomach is, as it were, pushed before them, and the œsophagus at the same time brought into being by the descent of the stomach. In point of fact, the descent of the œsophageal end of the stomach, and the consequent turning of the stomach with its left side foremost, may be described as the fundamental cause of the lesser sac of the peritoneum.<sup>2</sup> What has taken place in this foetus is, that at an early period, when the stimulation took place which produced the spina bifida, the spinal cord, the

<sup>1</sup> Virchow's *Archiv*, xlvii. 382.

<sup>2</sup> "The Peritoneum of the Human Subject illustrated by that of the Wombat," *Jour. Anat. and Phys.*, iv. 198, May 1870.

notochord and the commencement of the stomach became all firmly united, so that the stomach could not descend. But the thoracic viscera descending as usual, the diaphragm shut in the thorax below at the usual level, which happened to be just a little lower than the level to which the pylorus reached. Thus the narrow neck of the primary loop of intestine supplied by the superior mesenteric artery, the neck formed by the pylorus above, and the middle of the transverse colon below, came to be placed immediately above the diaphragm. Now, it is certain that the growth of this primary loop, as I pointed out many years ago, takes place in two stages,—the first a growth which, starting near the pylorus, forms the duodenum, and elongates the whole small intestine; the second, a growth from the colic end of the loop, which transfers the cæcum to its adult position. In this instance both ends of the loop were above the diaphragm, therefore both stages of growth went on within the thorax, and the condition of matters was brought about which was found. Also, the duodenum being developed within the thorax, and the coeliac axis coming off from the thoracic aorta, and the portal vein being fed from branches within the thorax, it naturally resulted that the portal fissure of the liver was pulled upon, and that the Spigelian lobe became visible within the thorax.

#### POST-ANAL DIMPLE.

In Specimens I. and VI. I have stated that there was a post-anal dimple. In both cases it was closely adherent to the tip of the coccyx. The depression was of a very distinct character, but expressed by the word dimple. Such a depression has, as Professor Turner kindly points out to me, been mentioned as an occasional occurrence by Luschka,<sup>1</sup> Hyrtl,<sup>2</sup> and Ecker,<sup>3</sup> and, according to Mr. Lawson Tait,<sup>4</sup> is by no means uncommon; but it is only recently that it has attracted my attention. I have

<sup>1</sup> *Anatomie des Menschlichen Beckens*, 1864, p. 57.

<sup>2</sup> *Topogr. Anat.*, 6th edition, p. 134.

<sup>3</sup> *Archiv. für Anthropologie*, 1879, xii. p. 138. Professor Turner also tells me that about twenty years ago he saw a well-marked post-anal dimple in an adult male, and has a cast of the region in his possession. He subsequently saw another example in a living male child shown to him by Dr. Arthur Gamgee.

<sup>4</sup> "On the Occurrence of a Sacral Dimple," *Report Brit. Assoc. Science*, 1878.

only seen one instance of a large and deep dimple in the adult. It was in a female subject, and the pelvis was preserved. It exhibits a sacrum of very remarkable appearance, there being but a slight longitudinal concavity on its anterior surface, while posteriorly the tip lies in a straight line with the extremities of the spines. It is  $4\frac{1}{2}$  inches broad at the base, and  $3\frac{3}{4}$  inches long, with a coccyx only half an inch long thoroughly united with it. In the anatomical collection in this University there is only one other example of straight sacrum. In it a rod laid along the front of the upper sacral vertebræ lies more than half an inch in front of the lower end, but there is a well-developed coccyx, and the length of the sacrum is  $5\frac{1}{4}$  inches.

Altogether, I have no doubt that the shortness of the sacrum from the subject with the dimple was the result of arrest of growth in the notochord, and that a well-marked dimple in the position described always results from that cause.

#### THE VARIETIES OF LUMBAR AND SACRAL SPINA BIFIDA.

Since writing these pages, Dr. Morton of this city, whose successful treatment of spina bifida is justly a subject of interest to the profession, has attracted my attention to the question of the position of nerves in cases coming before the surgeon; and with him I have looked at the specimens in William Hunter's collection in the University of Glasgow.

Of preparations from ten different cases, only two have the tumour completely covered with true skin. One of these (49a) is a tumour  $\frac{3}{4}$  inch in diameter. In it the cavity shows on the deep side a slight linear depression as if communicating with the cord which is situated in front; and on one side the deep wall is dissected away, showing the sacral nerves lying normally and coming from a level above the linear depression. The other skin-covered specimen (47) is large. It was unopened; but with the kind permission of Professor Young, the curator of the Hunterian Museum, I have opened into it, and find, as I expected, the cord in this instance also on the deep side of the dropsical cavity. This I expected, because I judged that such a tumour must be the result of delayed closure of the cord, or reopening of its canal behind at an early date. In the bottom

of the cavity there is a depression, in connection with which the cord seems to end; at the same time there is a communication with the arachnoid space.

Without entering into detailed description of the other specimens, it may be stated that they all of them present membrane of a non-cutaneous kind on part of their surface. In some of them the cord seems to end distinctly in connection with the superficial membrane. Some show the nerves of opposite sides coming off from the membranous surface crowded together in the middle line, there being evidently dropsy of the anterior sac of the arachnoid to such an extent as to lead to obliteration of the posterior sac. In others the nerves arise from the covering of the tumour in two series, which may be marked on the surface by punctiform depressions at their attachments, or which may spring from the lines of junction between a central membranous part of the tumour and two lateral skin-covered parts.

For practical purposes, spina bifida may be described as divisible into two kinds, the skin-covered and the membranous. The skin-covered are posterior to the spinal nerves; the membranous are traversed by nerves which arise from the membrane. But it ought to be understood that these nerves proceed to the intervertebral foramina, and have no connection with developed spinal cord.

## EXPLANATION OF PLATES XI. AND XII.

Fig. 1. Basal view of brain of Specimen III., about two-thirds of natural size. *a, a*, The portions of the hemispheres which were contained within the cranium, each exhibiting a dropsical hollow, the whole mass beneath the constriction lay outside the skull; *b, b*, elongated cranial nerves; *c*, medulla oblongata; *d*, enormously enlarged infundibulum with vestiges of optic commissure and nerves in front of it; *e, e*, dura mater partially removed from the hemispheres; *f, f*, vestiges of hemispheres of cerebellum.

Fig. 2. Cervical, dorsal, and four lumbar vertebræ of Specimen II.

Fig. 3. Skeleton of cleft palate, showing the arrangement of the vomer and intermaxillary bones of Specimen III.

Fig. 4. View of the displacement of viscera in Specimen IV. *a*, ear; *b*, symphysis of lower jaw; *c*, thyroid gland covering the larynx; *d, d*, lungs; *e*, heart; *f*, placed on the bodies of cervical vertebræ.

above the brachial plexus and the gap which separates the right from the left moieties of vertebral bodies and gives passage to the tissue which binds the stomach to the dura mater; *g*, stomach; *h*, spleen; *i*, vermiform appendage of cæcum; *k*, diaphragm; *l*, liver; *m*, umbilical cord; *n*, cord left by obliterated omphalo-mesenteric vessel (cord of Allen); *o*, rectum; *p*, urinary bladder; *q*, lobulus Spigelii; *r*, upper end of the obliterated omphalo-mesenteric vessel.

Fig. 5. Dorsal and four upper lumbar vertebræ of Specimen I.

Fig. 6. Portion of brain and spinal cord of Specimen I. *a*, corpora quadrigemina; *b*, *b*, hemispheres of cerebellum, with vallecule and vermiform process almost completely separating them; *c*, the extremity of the enormously elongated nodule; *d*, the recess in which the point of the nodule lay, and above it the opening of the central canal into the elongated 4th ventricle; *e*, the section of the spinal cord, showing a dilatation of the central canal which has been ripped open and found to end blindly without reaching up to the 4th ventricle.

Fig. 7. Cerebellum, medulla oblongata, and spinal cord of Specimen II, seen from behind. *a*, The lower end of the part of the cord flattened out in continuation of the 4th ventricle.

Fig. 8. Membranous area of spina bifida, and the tumour containing the brain, of Specimen IV. The tumour has been cut into by means of a transverse section; and the upper part, *a*, of its wall has been thrown forward from its natural position continuous with the cut edge *b*; below *a* is seen the falx cerebri; beside *b* is a foramen opening into the interior; *c*, *c*, right hemisphere partially cut across by the transverse section; *d*, left hemisphere a good deal injured; *e*, *e*, basal ganglia; *f*, membranous area over spina bifida; *g*, delicate membrane stretched across, limiting a dropsy of the 4th ventricle.

Fig. 9. Integuments, roots of nerves, and membranes removed from over the skull and spinal column of Specimen VI, and seen from the deep aspect. *a*, Basal view of the small hollow tumour representing the brain; and below *a*, the basilar artery bifurcating; *b*, a tough fibrous thickening of the dura mater where it projects forwards into the depression between the skull and the vertebral column; *c*, *c*, two strips of dura mater, from which emerge the right and left spinal nerves; *d*, anterior sac of the arachnoid, or that part which lies between the origins of nerves of the right and left sides; *e*, *e*, the right and left moieties of the posterior sac of the arachnoid; *f*, *f*, fifth cranial nerves.

Fig. 10. Magnified view of Specimen X., an embryo chick which has deviated from normal development about the end of the first day by exaggerated growth of the epiblast, which has become folded and frilled.

Fig. 11. Magnified view of a chick  $\frac{3}{4}$ -inch long, seen from the left. The upper aspect of the bare encephalon is seen, and at its back part the calamus scriptorius continuous with the central canal.

Fig. 12. View of the head of the same from the right, showing the common orifice of the mouth and ears, and the small cyclopiian eye.

**THE MINUTE STRUCTURE OF THE PALATINE NERVES OF THE FROG, AND THE TERMINATION OF NERVES IN BLOOD-VESSELS AND GLANDS.**  
By WILLIAM STIRLING, M.D., Sc.D., *Professor of the Institutes of Medicine*, and JAMES F. MACDONALD, *Student of Medicine in the University of Aberdeen.* (PLATE XIII.)

(From the Physiological Laboratory, University of Aberdeen.)

IN his *Leçons sur l'histologie du Système nerveux*,<sup>1</sup> Ranvier mentions that he uses the mucous membrane covering the hard palate of the frog as a convenient source for obtaining nerve fibres for the study of Henle's sheath. As this membrane is not attached to the bone which it covers, but is separated from it by a lymph sac, it is easy to detach the membrane and examine it. On doing so, what strikes one most of all is the particularly dense plexus of medullated nerve fibres which can be seen when its deep surface is examined. The following investigation was undertaken with a view to determine the arrangement and mode of termination of these nerve fibres.

*Arrangement of the Palatine Membrane.*—When the frog's mouth is opened the palatine arch is seen to be covered by a membrane, which is continuous behind with the mucous membrane of the œsophagus, while in front it is firmly attached to the maxillary and pre-maxillary bones along the line of the gums. Between it and the osseous lamellæ of the hard palate there exists a well-defined lymph space, so that, as remarked by Ranvier, when its connection with the œsophagus is severed, it is easily raised from behind forwards, a few touches with scissors being sufficient to divide the nerves and blood-vessels, which enter it near its posterior margin. In this region, near the posterior edge of the orbit, is the trigeminal foramen, which transmits the fifth nerve.

*Arrangement of the Nerves.*—The foramen lies between the prootic and transverse part of the parasphenoid, and is near the posterior and inner edge of the orbital cavity. Before it reaches

<sup>1</sup> Vol. i. p. 163.

this foramen the nerve has divided into three chief branches at the Gasserian ganglion, which pass through this foramen to their area of distribution. According to R. Wiedersheim<sup>1</sup> and De Watteville,<sup>2</sup> it is a purely sensory nerve, as no motor root has been discovered.

The three primary branches of the trigeminus are—(1) the *maxillary*; (2) the branch to the orbital and nasal cavity, or *orbito-nasal*; and (3) the *palatine*. Our observations refer more especially to the palatine branch. The palatine nerve, after passing out of the foramen, runs straight forwards on the deep surface of the palatine membrane, accompanied by an artery and a vein. It passes to the inner side of the levator bulbi. During its course it runs parallel with the longitudinal part of the parasphenoid. The nerves of each side are at equal distances from the middle line of the palate, and are parallel with each other. Opposite the point of the sphenoid the nerve divides into three branches of unequal size. The smallest is directed to the Harderian gland. This branch is divided when the membrane is isolated. The next branch has the original direction, and is intermediate in size; crossing the inner end of the palate bone, it passes through a foramen in the vomer. In its course it gives off branches. It then lies amongst the ducts of the inter-maxillary gland, which it supplies with a number of fine branches (Wiedersheim). One of its chief branches pierces the anterior border of the ethmoid cartilage, and reaches the "inter-maxillary space" and its glands, where it anastomoses with one or two fibres of the supra-maxillary branch of the trigeminus. According to De Watteville, after passing through the foramen in the vomer, it continues its course forwards and slightly outwards, joins a communicating branch of the orbito-nasal, and is distributed to the mucous membrane of the region. It is known as the vomerine branch.

The third branch, the maxillary, is the largest of the three. At the anterior end of the parasphenoid it makes a sharp bend outwards and forwards. It describes a semicircle round the anterior edge of the orbital cavity, passing along the anterior side of the palatine bone. It then disappears from the mem-

<sup>1</sup> *Die Anatomie des Frosches* (Ecker) Zweite Abth. p. 21.

<sup>2</sup> *Jour. of Anat. and Phys.*, vol. ix. p. 145.

brane. According to Wiedersheim, when near the junction of the palatine bone with the anterior end of the pterygoid, it pierces the fibrous membrane, between the bulbus oculi and the pterygoid process. Here it anastomoses with the superior maxillary branch of the trigeminus, and is distributed along with it to the skin of the upper lip and cheek. The above description is fundamentally the same as that given by Wiedersheim.<sup>1</sup>

*Arrangement of the Plexus of Nerves in the Palatine Membrane.*

—The general arrangement and ramifications of the palatine branches are seen in Plate XIII. fig. 1. The nerve and its chief branches lie on the deep surface of the membrane, but the finer branches pierce the substance of the membrane, to be distributed within it. At the posterior part of the membrane, where the nerves and vessels enter it (fig. 1, *a*), besides the palatine nerve proper, we have a number of other smaller nerves, which radiate from this point in several directions. These are mostly branches of the trigeminus, together with one small branch known as the palatine branch of the seventh nerve. This nerve passes outwards, and, according to Wiedersheim, it anastomoses with the branches of the trigeminus supplying the gums. From this centre (*a*) seven to eight small nerves radiate. These supply the posterior part of the palatine membrane. They give off very fine branches, which occasionally anastomose with each other. Those at the outer part anastomose with the branches of the palatine nerve which run back along the line of the gums, and also with twigs given off from the main trunk of the palatine nerve. Their ultimate ramifications are very fine, and even in a small area the microscope reveals a rich nervous supply. The greater number of the larger branches have a very serpentine course. This remarkable tortuosity is not obliterated by stretching the membrane, as may be seen when it is pinned out on a piece of cork. This tortuosity was noticed by Ranvier.

The palatine nerve proper, in passing forwards, gives off a great number of fine twigs, and also three or four larger branches. One of the latter arises shortly after the palatine nerve joins the membrane. This branch is of good size, and, running forwards in the same direction as the parent trunk, is chiefly distributed

<sup>1</sup> *Loc. cit.*



towards the middle line of the palate, and also externally in that part of the membrane lying upon the orbit. These smaller twigs anastomose with the branches coming from the parent trunk. A very fine plexus of fibres is thus formed in that part of the membrane over the orbit. This plexus communicates freely with small twigs from the recurrent part of the nerve upon the gums. After the chief nerve has turned outwards (fig. 1, *d*) along the anterior border of the palate bone, it gives off two large branches, which follow the course of the original trunk as far as the edge of the gums. The most anterior (*e*) of the three nerves sends one or two branches forwards along the gums, and then pierces the deeper parts. The middle branch (*f*) sends backwards one or two similar branches along the outer wall of the orbit, as far as its posterior angle, and then, piercing deeper parts, leaves the membrane. The posterior nerve (*g*) turns backwards into that part of the membrane overlying the orbit, and joins the fine plexus in this part. This plexus is the most striking part of the nerve supply. It is shown in fig. 1 (at *x-x*). It is formed of anastomosing and branching fibres. Some of the fibres of one branch join a neighbouring branch. Some of them appear to be true recurrent nerves, for, after anastomosing, in many cases their direction changes, and, instead of following a peripheral course, they seem to return upon themselves, and form true recurrent fibres.

The nerve-supply on the two sides of the same palate corresponds exactly even to minute details. The number and arrangement of the nerves is similar in both. If we compare the palatine membrane of several frogs, we may see that the mode of nerve-distribution is quite similar, and that they correspond almost exactly in the final distribution of the nerves.

*Method of Preparation.*—In order to see clearly the coarser plexus just described, the palatine membrane is prepared in the following way:—The frog's head having been cut off, so as to include the whole mucous membrane of the hard palate and pharynx, and the lower jaw being removed, the head is now put into a solution of chromic and nitric acids, and left in it for about ten days, the fluid being renewed once or twice. In ten days the membrane has become somewhat hard and quite translucent. Before mounting it the epithelium covering it should be removed

gently by a scalpel, in order to increase the clearness of the object. It is then washed in water, and, after the acids have been removed by sufficient immersion in water, it is mounted in glycerine, whereby the plexus is rendered quite distinct. A better plan is to employ the fluid used by Freud,<sup>1</sup> consisting of a mixture of water, glycerine, and nitric acid, containing nitrous acid. In two or three days this method renders the membrane more transparent than the above method, and the epithelium is easily removed by scraping. The nerves may be fixed by subsequent immersion for several hours in a 1 per cent. solution of osmic acid.

Again, the plexus may be shown by "fixing" the elements of the membrane by osmic acid solution (2 per cent.), having previously pinned it out on a piece of cork. When the nerves have been sufficiently acted on—i.e., in from 10 to 15 minutes—the specimen is removed from the acid, washed with water, and mounted in glycerine.

*Arrangement of the Blood-Vessels.*—The general arrangement of the blood-vessels corresponds closely with that of the nerves. The chief artery and vein of the palate are arranged beside the palatine nerve. Their branches are given laterally in much the same order as the branches of the nerve. At the front end of the parasphenoid, where the nerve breaks up into its primary branches, the artery runs forwards with the vomerine branch. Then it gets into the region of the ducts of the intermaxillary gland, and it gives off a number of large branches which soon break up into a plexus of smaller vessels which anastomose very freely. At other parts of this membrane there is also to be found free arterial anastomoses. All these vessels are situated on the deeper surface of the membrane, but as they become smaller in size, they penetrate more towards the buccal surface. At parts a fine capillary network appears just under the buccal surface. The meshes of this network are very regular, leaving quadrilateral spaces between them. After staining with picrocarmine, the structure of the arteries and veins comes out very distinctly. In the tunica adventitia a regular series of stellate pigment cells is to be seen. These cells are more thickly set the larger the

<sup>1</sup> W. Stirling, *Text-Book of Histology*, 1882, p. 84.

calibre of the vessel becomes, and they are more numerous around the arteries than the veins.

*Plexus of Medullated Nerve Fibres.*—The rich plexus of nerves which exists on the deep surface of the palatine mucous membrane consists chiefly, but not exclusively, of medullated nerve fibres. If thin shreds of the deeper layers of the membrane be torn off after exposure to osmic acid, and if the shreds be stained with picrocarmine (twenty-four hours) we obtain a good view of the arrangement and nature of the fibres. We discover that a large number of *ganglion cells* lie scattered amongst the fibres, and they occur in groups of two, three, or more, chiefly where a division of the nerve takes place, although they are by no means confined to the points of division. A nerve trunk may contain ten to fifteen or more medullated nerve fibres, which are enclosed in a sheath of Henle provided as usual with an endothelial lining. The nerves sometimes change their relations within the sheath. A few non-medullated fibres exist along with these fibres. Between the sheath and the included fibres there exists a space, which is to be regarded as a lymph-space.

These larger nerves may divide into two or three smaller bundles, or they may sometimes give off branches laterally containing two to six nerve fibres. These small bundles subdivide still further, until there are only one or two fibres running together. Ultimately the nerves are composed of single medullated fibres. The course of these branches is by no means in the direction of the parent trunk, but is in the form of a plexus or network. Sometimes the branches anastomose, but generally they cross each other's path. This crossing takes place at different planes in the membrane, so that the fibres do not come into contact. If vertical sections of the membrane are examined, the arrangement may be readily made out. The palatine nerve and its largest branches run along just under the deep surface of the membrane, being attached to this surface by fibres of connective tissue. The larger branches lie between the deeper planes of the membrane, but on coming nearer to the buccal surface, the size of the nerve bundles becomes less. In vertical sections of the palate they are often cut obliquely, while at other times they are cut longitudinally.

In all these bundles it is very evident that the sheath does not lie closely on the nerve fibres, but a small space intervenes between them. As pointed out by Stricker, this is no doubt a lymph-space. A similar lymph-space was observed by Klein,<sup>1</sup> in the nerve plexus of the nictitating membrane of the frog. At the point of division of the bundles it is often evident that besides a simple separation of the fibres, a true *division* of some of the nerve fibres into two fibres takes place. This is well seen in the small bundles containing two to four nerve fibres, as we may count a greater number of fibres in the branches than in the parent trunk. At the planes of division of the trunks the sheath of Henle on the branches becomes continuous with that on the trunk.

*Arrangement and Structure of the Ganglion Cells.*—As already mentioned, numerous *ganglion cells* occur in connection with this plexus (Pl. XIII. fig. 2). These ganglion cells are frequently situated at the point of division of the bundles, but they may be also found at other parts. They are not arranged in large groups, but are placed at intervals along the course of the fibres, while two, three, or more cells may be situate near each other at the point of division of the bundles, where they are most numerous. They are always enclosed in Henle's sheath—(in addition to their own capsule)—which is slightly raised opposite where they occur. Nor does the sheath lie directly upon them, being separated from them by its endothelial lining. If we examine the cell closely with a No. 8 lens, and with the tube of the microscope drawn out, we readily make out the following structure:—The cell has an ovoid shape, with a well-defined capsule, which is lined by endothelial cells whose nuclei are apparent, seen either on edge or *en face* within the capsule; the body of the cell consists of so-called "granular" protoplasm. Towards the narrow end of the cell the protoplasm is continued outwards as a *straight fibre* or process. This straight process passes into the nerve bundle, and sometimes it seems to join a non-medullated fibre. Axel Key and Retzius represent the sympathetic nerve cells of the frog with the spiral fibre continuous with a medullated fibre, while the straight fibre remains non-medullated. The broader end of the cell is composed of a large mass of granular

<sup>1</sup> *Quarterly Journal of Micros. Science*, vol. xii. p. 23.

protoplasm filling up the capsule, but where the straight process comes off, the cell wall becomes more distinct from the protoplasm, and a considerable space intervenes. By altering the focus upwards or downwards a little, it is easily made out that this space is occupied by a fibre which winds *spirally* round the straight process, the *spiral* fibre of Beale and Arnold. This fibre is finer than the straight process. The coils of the spiral become closer, and the diameter of the coil greater as it encroaches more and more on the protoplasm of the cell. This spiral surrounds at least one-third of the protoplasm of the cell. The peripheral end of this spiral process runs along with the straight process into the nerve bundle, and both are surrounded with the capsule which forms a sheath for them. Ranvier<sup>1</sup> has figured the spiral process as piercing the sheath, and coming into relation with the straight process of the ganglion cell. His description applies to the ganglion cells found on the vagus ganglion of the frog. We have not been able to make out the same arrangement in these ganglion cells. At the larger end of the cell, placed eccentrically in the mass of protoplasm, is a spherical nucleus, with a nuclear envelope, and the nucleus contains a small well-defined nucleolus. This nucleus is situated at the end of the cell furthest away from the straight process. There was no evidence of any connection between it and either of the processes. These cells are in fact practically the same as those that occur in the sympathetic system of the frog, and which have been described in other parts of the frog's body by many observers. It would therefore seem that these cells have a very wide distribution, for they occur in the sympathetic system, the heart, the vagal ganglia (Ranvier),<sup>2</sup> and in the lung (Arnold,<sup>3</sup> and W. Stirling).<sup>4</sup>

*Plexus of Non-Medullated Fibres.*—Before describing the dense network of non-medullated fibres, a short account of the structure of the palatine membrane is necessary. In transverse sections of specimens hardened in chromic and nitric acid solutions, and stained with picrocarmine, or of those prepared by the gold

<sup>1</sup> *Traité technique d'Histologie*, p. 842.

<sup>2</sup> *Traité technique d'Histologie*, p. 842.

<sup>3</sup> *Virchow's Archiv*, xxviii. 1868.

<sup>4</sup> Read at the Royal Society, 16th Dec. 1882

chloride method, the structure is very well seen. On the whole, it has a striking resemblance to that of the cornea—fibrous lamellæ and stellate branched cell-spaces. In the deeper parts of the membrane the texture is looser. Here the cell-spaces are fusiform and less ramified, and the fibrous nature of the lamellæ is very distinct. To see this fibrous matrix more readily, a transverse section should be well teased. The fibres lie in the same horizontal plane generally parallel to each other, and do not interlace much with those above or below them. The spaces seem at first sight to be branched cells, as they have a dark outline, especially in the gold preparations, and deeply-stained corpuscles are found to occupy the spaces exactly as in the cornea. As we approach the buccal surface of the membrane the texture becomes denser; the spaces are more stellate and ramified, and their processes join those of similar spaces above and below them. Still the layers are generally noticed to be parallel with the buccal surface. Just under the epithelium there is a rich capillary plexus seen in section. The epithelium on the surface consists of oval or columnar cells, with cilia on their peripheral end. Their central end is narrowed, and fits in between a layer of smaller cells lying on the surface of the membrane. These smaller cells are apparently in process of development, as other cells intermediate in size between these and the fully developed ciliated cell may be found. The fully developed cell is oval, and has clear contents, with an oval nucleus at the lower end. Its central end, as mentioned above, is set on the surface of the membrane; its peripheral end is covered by a thick clear band, and through this the cilia pass out. This thickened band or cuticle overlies all the cells of the membrane, with the exception of the goblet cells, which are numerous. Between these epithelial cells lie a great number of goblet cells. The characters of both kinds of cells have been described by Professor Michael Foster in the *Journal*.

Between this layer of cells and the fibrous tissue there is no distinct limiting or basement membrane. To make out the plexus of nerve fibrils gold preparations are alone serviceable. If thin sections be shaved from either surface of the membrane, and examined with a No. 8 lens, a beautiful network is at once revealed. This network is not quite so rich as that of the cornea.

If we trace a non-medullated fibre for a short distance we may see it bifurcate, and one or both of the branches ends in a bundle of fibrils. Such a network is shown in Pl. XIII. fig. 4.

The bundles of fibrils pass off in various directions, becoming finer and finer by repeated division. Very often, at the place where the myeline sheath ceases, there is an irregular swelling on the nerve, and from this the fibrils pass. Again, along the course of these latter there are little granules or swellings of various shape. Some of them are oval and clear, and are several times the thickness of the fibril (Pl. XIII. fig. 6).

The larger divisions of the axial cylinder may be traced until they join similar branches from other nerves. Sometimes they end in an irregular mass of protoplasm, with which several larger and smaller fibrils are connected. These masses have an ill-defined outline, and are usually dark and deeply stained. At times an oval body, resembling a nucleus, is to be seen in their centre. The great majority of the terminal fibrils join the meshes of the irregular plexus found throughout the whole membrane. This latter, for sake of clearness, we shall call the *ground plexus*. This *ground plexus* consists of fine threads of tolerably uniform diameter. They lie mostly parallel with the surfaces of the membrane. They do not anastomose very often, thus leaving large meshes between them. The points of union of two or more fibrils are marked by triangular and irregular dark bodies or corpuscles, which are striated in some places. Four or five threads may be seen running parallel to each other on the same plane. Again, the threads may have a beaded outline. Some of them also pass into deeper or more superficial planes; thus throughout the membrane there is a connected network of fibrils. When the focus is changed a new series of fibrils on a deeper or higher plane is revealed. The fibrils are more numerous near the buccal surface. Here the fibrils have a straight course; they do not anastomose quite so freely, and their diameter is smaller. There is no evident connection between the threads of this plexus and the cell-spaces or cells of the membrane. Klein<sup>1</sup> has described a marked relation of the non-medullated nerve plexus to the pigment cells in the membrana nictitans of the frog. There are similar pigment

<sup>1</sup> *Loc. cit.*, p. 29.

cells in the palatine membrane; but we have not been able to make out any further relation between them and the plexus just described, than the fact that opposite these cells the nerve fibrillæ often bend in their course, and pass before or behind them.

On transverse section it is seen that the medullated nerves run up sometimes almost quite as far as the buccal surface, while sections of them may be found at various levels in the membrane. Thus it would appear that the medullated nerves pass into the non-medullated plexus at all parts, except perhaps just under the buccal surface. The arrangement does not seem to be that the medullated nerves are confined to the deeper parts of the membrane, and their axial cylinders transmitted onwards as a non-medullated plexus. The fibres cannot be followed continuously for any distance. They appear rather as short threads scattered throughout the section. The greater number of these are directed vertically towards the buccal surface, although in thicker sections others may be found with a more horizontal course. When the section is examined carefully near the buccal surface, more fibrils are to be seen than is evident at first on a superficial examination. All these are directed towards the epithelium, and may be seen at parts to pass clear beyond the fibrous matrix into the layer of epithelial cells. We have found it impossible to trace these fibres in thick sections, as the epithelium strikes a dark colour with the gold, and so the outline of the fibril is lost. In some very thin sections, where there is at most a single layer of cells, the part is then more transparent. In such sections we have seen very fine fibrils passing in between the cells, just as similar fibres pass into the thicker layer of epithelium on the anterior surface of the cornea; but our observations do not enable us to say whether these fibres, which pass up between the epithelial cells end in free fine points, or form a plexus lying between the epithelial cells.

*Termination of the Nerves in Relation with the Blood-Vessels, Arteries, and Capillaries.*—In portions of the palatine mucous membrane which have been stained with one per cent. gold chloride solution for an hour, and afterwards reduced by the formic acid method of Ranvier,<sup>1</sup> admirable views of the relations of the

<sup>1</sup> W. Stirling, *Text-Book of Physiology*, p. xlv.



nerves to the arteries, and the nerve fibrils to the capillaries may be obtained. In Pl. XIII. fig. 3 the nerve-supply of a small artery is shown. If a small artery be examined, it is noticed that at intervals, which vary in length, medullated nerve fibres run on to it. A single fibre passes off from a neighbouring bundle of nerves, and enters the tunica adventitia of the vessel. Or a medullated fibre, winding through the neighbouring tissues, when near the artery may divide into two, one of the branches going to the vessel, the other keeping the course of the fibre before division. When these medullated fibres enter the vascular wall, they do not at once lose their sheath, but run upwards or downwards on it a short way, and may meet with other similar fibres. This may be called the outer plexus.

Soon those medullated fibres begin to break up. They give off irregularly a series of branches, which join the plexus of fine fibres lying beneath them, or sometimes these branches form a few loops of a secondary plexus. At the part where they arise from the medullated fibre, there is generally a swelling on the parent fibre, which is stained black by the gold chloride. Again, along the fibres which arise there, bead-like swellings occur, some larger some smaller, giving the fibres irregular outlines. The swelling, which is often seen at the plane of division of the medullated nerve, is probably ganglionic. This may be called the middle plexus.

If we examine the vessel wall at any part where the chloride of gold has acted efficiently, we easily notice a beautiful meshwork of fine nerve fibrillæ. As a rule the interstices of this meshwork are very regular, leaving spaces between, which are often quadrilateral. Beads or small swellings are found at the nodes of the network and also on other parts of the fibres, especially where the fibrillæ of the middle plexus join them. This plexus appears on same level as the muscle fibre. Connected with the meshes of this plexus we may notice at intervals triangular or irregular dark masses. Sometimes these are clear in the centre with a dark outline. Possibly these are ganglionic cells in the wall of the artery; at least they are all connected to the neighbouring meshwork, and very often fibres of the middle plexus enter them. From this, which may be called the inner plexus, it is most probable that fine threads are

given off, which end directly in the muscle fibres of the middle coat. In some parts of the specimen some such faint threads could be seen with a No. 9 lens. But still this observation has not been clearly proved. The nerve endings on the veins are similar to those on the arteries. The outer and middle plexus is not marked, and fewer medullated nerves reach the veins. The inner plexus is formed apparently of much finer fibres, and the meshwork is not so dense, leaving larger interstices.

The capillaries differ somewhat from the arteries and veins in the mode in which the nerve ends on them. Here we have not the same plexiform arrangement found in the larger vessels. The fibrillæ all run longitudinally and parallel with the vessel, but not quite close to it (fig. 5, *nc*, *nc*). There are generally two fibrillæ along the side of each vessel, but there may be more in some places. They are easily seen, as the capillary wall is quite transparent. At some places they anastomose with each other, and where one branch of the capillary joins another, there the nerve fibrillæ anastomose. They do not always keep the same relative position on the capillary, but wind round it apparently in a long spiral. These nerve fibrillæ do not appear linear, but look sometimes like a string of beads, the fibres being finely marked with little dots or swellings. At parts there are fibres which lie upon the wall of the capillary, but as a rule they do not touch the wall of the vessel. Again, they do not appear to form any anatomical connection with the nerve fibrillæ of neighbouring parts.

When this article was being prepared for the press, we noticed in the October number (1882) of the *Archiv für Microscop. Anatomie*, a paper by Bremer, on the nerve endings in blood-vessels. He has made use of the vessels in the striped muscles of the frog and lizard as objects of investigation. The results he obtained are quite similar to those we have described. He thus describes the ending of the nerves in the capillaries:— In good gold preparations one or two still finer nerve fibrillæ are to be seen closely attached to the capillary, and by means of many small knot-like thickenings are related to the capillary wall. These thickenings, which look on profile like small outgrowths of the nerve fibrillæ, are the ultimate endings of the capillary nerves. They do not anastomose with the nerves of

the neighbouring tissue, nor do they give off branches to the same, but form a completely separate system. The only exception to this rule is, that sometimes the capillary nerves are in relation with the nerve ending in the striped muscles. The nerve endings have nothing to do with the nuclei of the capillary wall.

*Nerve Endings in Glands.*—We have already seen that the anterior part of the palatine membrane, viz., that part in front of the parasphenoid, is thickly beset with glands and gland ducts. If we separate a thin layer from this part in a specimen carefully prepared by the chloride of gold method, we may be successful in getting a preparation showing the relation of glands, capillaries, and nerves. A drawing of such a preparation is given in Pl. XIII. fig. 5. These glands are not set closely together, but considerable spaces intervene. They are situated nearer the superficial than the deep surface of the membrane. To the naked eye they appear as numerous points studding the specimen. Examined with the No. 7 lens they are dark masses, in which the cellular structure is almost obscured by the deep staining of the reagent. Around their edges the outline of the cells can be made out distinctly; and if, in the process of preparation, one of them has been squeezed, the cells are spread out and become distinctly visible. The outline of these cells is mostly polygonal, some may approach the triangular form, others are oval. On side view their appearance is similar; so they are cubical gland cells.

In the intervals between these glands we have the capillary vessels. These capillaries form a network with the glands in the meshes. Upon these capillaries a beautiful plexus of nerve fibrils is found as already described. Sometimes it is very rich, four fibrillæ running parallel with the vessel. These fibrillæ are not all of the same size, one at least being larger and running more directly. The smaller cross the vessel oftener, and anastomose more frequently. From these larger fibres branches come off at certain parts, and proceed to the mass of gland cells. Sometimes such a branch arises from a triangular swelling on the parent fibril, which at this point breaks up into two, one going to the gland, the other continuing its course along a neighbouring capillary. These branches are on the same plane as the wall of the capillary vessels, being in full focus at the

same time. They are thus, without doubt, the proper nerves of the capillary.

Those branches generally run to the gland in the same plane as they left the capillary. Before reaching the gland acinus they give off branches at intervals, the original fibre passing half-way round the gland and the branches striking into it. But this arrangement is not constant. Sometimes the fibre does not branch before entering the gland. Again, it may branch and anastomose with branches of neighbouring fibres. We have not noticed any such plexus enveloping the gland, as Klein<sup>1</sup> described in the nictitating membrane of the frog. The farther distribution of these nerves in the gland could not be traced, so that we are unable to say whether the nerve fibres which enter the gland in relation with its secretory cells actually terminate within the secretory epithelial cells, as described by Pflüger for the sub-maxillary gland of the rabbit, and Kupfer for the salivary glands of the cockroach. Otto Drasch<sup>2</sup> has described a somewhat similar relation of nerve fibrils which pass from Meissner's plexus to the acini of Brunner's glands.

### DESCRIPTION OF PLATE XIII.

Fig. 1. This shows the general arrangement of the coarser bundles of nerve fibres as they occur on the deep surface of the palatine membrane. The membrane is supposed to be transparent to show the nerve fibres on its deep surface. *xx* is the portion of the membrane over the bulbus oculi; *a* is where the nerves come out at the trigeminal foramen. Hartnack, Oc. 3, Obj. 3 (reduced).

Fig. 2. Small branch of the "medullary plexus" isolated. The dark fibres are the medullated fibres. The fibres are invested by Henle's sheath (*h*). Along the course of the fibres, but under the sheath are nerve cells *cc*. Oc. 3, Obj. 9 (reduced).

Fig. 3. A small artery with the nerves distributed to it. *m* = medullated fibre, and *p. f.* the plexus of nerve-fibrils with little swellings where they join, in relation with the muscular or middle coat. Oc. 3, Obj. 8.

Fig. 4. Plexuses of nerves seen in palatine membrane. The various nerves lie on different planes. *m* = medullated nerve fibres, *f* = fibrils, and *g* = swellings (ganglionic?) lying at their junctions. Oc. 3, Obj. 8.

Fig. 5. Distribution of nerve fibrils (*nc*, *nc*) to capillaries *CC*, and also (*ng*) to glands *GG*. Oc. 3, Obj. 7.

Fig. 6. Termination of a medullated nerve *m* in a plexus of fibrils *f f*. Oc. 3, Obj. 8.

<sup>1</sup> *Loc. cit.*, xii. p. 27.

*Sitzungsber. d. k. Akad. d. Wissensch.*, Bd. 82, Abth. 3, p. 168.

THE LYMPHATICS OF PERIOSTEUM. By GEORGE HOGGAN, M.B. (Edin.), and FRANCES ELIZABETH HOGGAN, M.D. (Zurich), M.K.Q.C.P. Ireland. (PLATE XIV.)

IF the suggestion which we made in our last paper on the lymphatics of vascular walls be correct,—that one of the functions of the lymphatic system is to afford to injured tissues a ready supply of reparative material,—then certainly, judging from the amount of plastic lymph which becomes effused round a fracture of bone, and which all the later surgical writers describe as being poured out from the periosteum, that membrane or organ must be provided with lymph vessels. On the other hand, with one exception, all the later investigators of the lymphatic system either deny or ignore the existence of lymphatics within the periosteum; and it only requires a glance at the one excellent drawing afforded by the one exceptional investigator, to assure us that the vessels which he supposed to be lymphatics are nothing else than the distinctive blood-capillaries of the tissue in question. It was the consideration of the foregoing facts that first impelled us to search for, by means of our special methods, and to discover, with very little difficulty, the lymphatics of the periosteum which we are about to describe.

The actual state of scientific opinion upon this question will be best shown by some extracts from the published works of the few investigators who have referred to it; and, in view of what we have already said, we shall also include all speculations on the existence of lymphatics of bone, as being inseparable from the question of the existence of the lymphatics of periosteum. We may, at the same time, give it as our opinion that, apart from the existence of the periosteum, bone possesses no lymphatics, and that in the periosteum itself the lymphatics seem specially situated where they can have no direct contact with bone. Cruickshanks<sup>1</sup> is the first investigator who states that he had seen lymphatics in bone, he having injected lymphatics coming out (*sic*) of the body of one of the dorsal vertebræ, in the

<sup>1</sup> *Anatomy of Lymphatic Vessels*, 1780, p. 198.

substance of which he also saw them ramifying. This statement appears to be stereotyped in all subsequent anatomical works; but, with Sappey, we believe it to be of no value, and we further say that even if they had existed as described, it would be impossible either to inject them *backwards* or to follow them out in the hard bony tissue, as described by Cruickshanks, by any method hitherto devised. With reference to this statement of Cruickshanks, as well as to those subsequently made by Brugmans, Breschet, and Bonamy, Professor Sappey<sup>1</sup> remarks:—

“Breschet cites a note of M. Bonamy, in which that author announces that he had found lymphatics coming out of some holes in the inner condyle of the femur, and which he had even followed to some depth through the spongy tissue. I have sought to control all these assertions, and with this intention I have undertaken some rather extensive researches. The results I obtained were always negative. After having again attentively read the observations recorded by all the above-mentioned authors, and some other authors which it would be needless to mention, I have no hesitation in declaring that not one of them can be accepted as conclusive. In reality they are of no value. I remain convinced that, up to the present time, no anatomist has seen lymphatics in osseous tissue, and I am also convinced that none exist there.”

While agreeing entirely with Sappey in the foregoing opinions, we of course differ from what he says of the periosteum, which is as follows, page 11:—

“Ligaments, aponeuroses, tendons, the periosteum, the dura mater, the sclerotic, the fibrous envelope of the corpora cavernosa, all the fibrous tissues, in fact, are destitute of lymphatics.”

Cruveilhier,<sup>2</sup> another French investigator of the lymphatic system, states:—

“No one has yet demonstrated the existence of the lymphatic vessels in bones, but it is probable that they exist there, as the phenomena of nutrition and certain morbid phenomena tend to prove their existence.”

Again, at page 128, he says:—

“As for the lymphatic plexuses which might exist in the cartilaginous and bony tissues, I have never been able to discover any.”

Apart, however, from opinions founded on personal investiga-

<sup>1</sup> *Vaisseaux Lymphatique*, 1875, pp. 11, 12.

<sup>2</sup> *Anatomie Descriptive*, 1852, p. 47.

tion, we have the mere opinions of many great anatomists for or against the existence of lymphatics in bone, but it will be observed that, with the exception of Sappey, they make no specific mention of periosteum, which they seem to include under the head of bony tissues. Of the more important anatomists, Mascagni believed in the existence of lymphatics in bone; Kölliker doubts it, while John Hunter,<sup>1</sup> whose vagaries on the question of absorption by the lymphatics have had, and still have, such a disastrous effect on scientific opinion in this country, not only was certain of the existence of lymphatics in bone, as everywhere else, but he specifically states, over and over again, that, by means of vivisectional experiments, he had been able to demonstrate their absorbing and modelling action upon the bones, and at page 257 he even goes the length of speculating on the kind of mouths which the lymphatics of bone must possess to enable them to absorb such hard material, and suggests—

*“That if we could see the mouths of these vessels, we might perhaps class the modes of absorption, as animals have been classed, by their teeth.”*

Unfortunately for Hunter's opinions and the results of his experiments, the actual anatomical facts show that, even in the periosteum, the lymphatics not only do not come in contact with the surface of the bone, but that they are, on the contrary, situated as far as possible from it.

We now come to the one exceptional anatomist, already referred to, who professes to have discovered the lymphatics of the periosteum. In the thirteenth volume of the *Archiv für Mikroskopische Anatomie* for 1877, p. 87, Dr. Albrecht Budge contributes an article entitled “Die Lymphwurzeln der Knochen,” or radicles of the lymphatics in bone. It is illustrated by five figures, the three of them drawn by himself being *schematische*, following that habit so dear to the German scientific soul of putting one's hypothesis in a drawing as well as in writing. The other two drawings are by Herr Weiland, the drawing-master of the University of Greifswald, and they leave nothing to be desired in the matter of accuracy. The first drawing is from a silver preparation of the inner surface of the periosteum, and purports

<sup>1</sup> Surgical Works of John Hunter by Palmer, 1835, vol. i. p. 254 *et alia*.

to show the lymphatics of the periosteum on that surface; and that accurate drawing enables us to state, without a moment's hesitation, that what Budge has described as lymphatics are nothing other than the blood-capillaries of the inner surface of the periosteum, where, moreover, as a matter of fact, the lymphatics never exist. In the drawing the lymphatics are white and the blood-vessels blue, and the drawing-master, with unconscious fidelity, has drawn the blue vessels in direct continuation with the white lymphatics. It is even surprising that an inspection of the drawing did not warn Dr. Budge of the mistake he was committing in describing as lymphatics the ordinary and distinctive blood-capillaries of the inner surface of the periosteum (see our fig. 6). The explanation of the mistake, however, is easy; it is evidently due to a cause that has led many into error. We ourselves, when we first employed silver imbibition, were led into a similar error, which may therefore be now explained to prevent others falling into it in future. In Dr. Budge's preparation he had first injected the blood-vessels with gelatine coloured with Berlin blue. But when capillaries injected with this material are exposed to the action of nitrate of silver, the capillaries are bleached, and according to the relative proximity of such injected capillaries to a surface, as well as the relative thickness of the coats of arteries or veins, so do we find the bleaching action to vary. That some portions of the capillary plexus retained their blue colour no doubt led Dr. Budge to hold them to be the whole of the blood-vessels of the part, and to imagine that therefore the white, that is the bleached portions, must necessarily be lymphatics. The actual relations of the lymphatics on the outer surface of the periosteum with the blood-capillaries on the inner surface is shown in our fig. 6. The blood-capillaries, *c, c*, shown there are identical with those shown in the same locality by Budge as lymphatics. The branched cells lying alongside of the capillaries in both drawings may be taken as the gauge of the respective scales of the drawings. The second figure (fig. 4), drawn by Weiland from nature, refers to the forcible driving of blue injection into the cell-cavities of bone, a fruitful source of error, which has already been explained in our article on the lymphatics of the pancreas in this *Journal*.<sup>1</sup> In short, Dr.

<sup>1</sup> July, 1881.



Budge's conclusions, from beginning to end, are erroneous, and his textual descriptions so vague that, but for the clearness and accuracy of the two figures referred to, it would have been difficult to understand what he meant, or to control so completely as we have been enabled to do the errors he has fallen into.

We regret to have felt it imperative to enter at such length into Dr. Budge's supposed discovery. It is always so much easier to describe discoveries where previously all was acknowledged to be unknown, for rival claims and contrary descriptions leave those uninitiated into special methods and special researches very much at a loss to discover which is right or which is wrong.

The principal method of preparation employed by us is the one already described by us on several occasions in these pages, by means of silver imbibition of both surfaces of the periosteum, after it has been mounted upon our histological rings. For convenience's sake, we generally choose the larger long bones of the larger mammals, including man. We also choose only those portions of periosteum to which no muscle, tendon, or aponeurosis is attached, lest any arrangements of lymphatics found there should be held to be modified by the attachment of those structures. Such portions are easily found on the tibia and humerus; in the former, indeed, portions of the periosteum may be found, whose attachment to the bone is so very slight, that they can be stripped off without doing any material damage to the inner surface of the membrane. As in other cases, the lymphatics can always be most easily shown in lean animals; but it is also advisable here to examine the lymphatics in the periosteum of fat animals (see fig. 5), so as to observe the displacing action of the fat cells on the larger and more superficial lymphatics. Care, however, must always be exercised in such cases not to break up the fat tissue on such surfaces, or to touch them with greasy fingers, which prevents the silver solution from penetrating among and defining the various elements which compose, or are included in, the periosteum. After the muscles and any loose tissue have been dissected off the outer surface of the periosteum, a slit is made in a favourable spot, and the membrane seized with a pair of broad pointed forceps. It is thus firmly held while it is being gently torn off the

subjacent bone, any slight adhesions being divided by the point of the scalpel. In this way a portion of the sheet, large enough to be mounted on a pair of our rings, is easily obtained, and the subsequent steps are similar to those we have so often described.

In addition to the foregoing method, we have lately used another controlling method, both upon the periosteum and upon other tissues which we have previously investigated. Having first, by means of silver imbibition, acquired a familiar knowledge of the lymphatics in the tissue, and their general position, relations, and the directions of the lymph flow, we take a hypodermic syringe, furnished with a very fine needle canula, and inject the lymphatics with a one-tenth per cent. solution of nitrate of silver in distilled water. In the case of the periosteum, great care must be used in inserting the needle, on account of the thinness of the membrane, for if the point of the needle passes through either surface, failure at that point is certain. For this method the periosteum is left attached and undisturbed upon the bone, and it is fortunate if, after a dozen of trials, one at length succeeds in getting into the lymphatics with the silver solution. When successful there is no difficulty in noticing it, and on such occasions we place our finger nail at the point where the lymphatic is leaving the periosteum, and where, consequently, the silver solution is seen to be running out; by compressing the lymphatic there we are able to bring a considerable pressure upon the interior of the lymphatics. The great advantage thus obtained will be afterwards explained in connection with what we have to say about lymph radicles. Apart from this, and except perhaps in tracing connections between two surfaces in a thick tissue, the method is of no great advantage. The silver stream follows the shortest course towards exit, and even when pressure is made, as already explained, the branches which form junctions with the main stream close their valves and prevent the silver solution passing up. The consequence of this is, that in a tissue like the periosteum, where the afferent and efferent lymphatics are combined, one only sees the main stream, and closely contiguous channels are undemonstrated. For this reason all our figures are drawn from preparations of silver imbibition of surfaces, where every vessel and every cell or other element is clearly shown.

When a number of successful preparations have been thus made from the periosteum of different animals, both surfaces of the membrane having been stained in every case, the first prominent and invariable feature which presents itself is, that the lymphatics are to be found plentifully, in every case, on the external surface of the membrane, but in no case whatever, in our experience, on the internal surface, or that lying next to the bone. Considerable differences will also be seen to exist in the arrangement of the lymphatics on the outer surface in different specimens, but these differences are due principally to the unequal thickness of the membrane at different points of the same bone, at different ages of the same animal, and in animals of different races. As, indeed, the varying structure of the membrane itself, now well recognised, is the principal factor in the peculiarities seen in the lymphatics, to understand these peculiarities it is necessary to have a precise idea of the general structure of the periosteum, and of the changes it may undergo under the influence of certain conditions.

At first sight it would seem that a precise knowledge of so simple an organ as the periosteum was not so very difficult to obtain. This, indeed, ought to be the case; but if we follow the teachings of various schools, and the description of various histological investigators, the greatest possible diversity of opinion is found actually to exist. Professor Kölliker (and following him we have both the German and English anatomical schools) describes the periosteum as a fibrous membrane, of which the outer layer consists of white fibrous tissue, containing occasionally fat cells, while the inner layer is made up of elastic fibres, having the appearance of being composed of several strata of elastic membrane. On the other hand, Professor Charles Robin (and following him we have the French school) asserts even vehemently that the periosteum is not fibrous, but formed of cellular tissue. It would seem that while the one only examined the periosteum in young animals, the other only examined it in old ones, that being the only explanation available, while as a matter of fact the truth seems to lie midway between them. In fig. 6, from the humerus of an old horse, the cellular elements on the inner surface of the periosteum are at their minimum, and fibrous or gelatinous tissue seems to compose the whole of

the membrane, while in fig. 2, from the tibia of a goat scarcely full grown, the cellular element greatly predominated, and, as seen on comparing figs. 1 and 2, considerable modification in the appearance of the lymphatics is one of the prominent results. On examining the inner surface of the portion of periosteum from which fig. 2 was drawn, it was seen to be composed of a dense mass of the ordinary branched cells of the part, embedded in the ordinary clear gelatinous matrix, which, however, was exceedingly scanty in comparison with the immense numbers of the cells it contained, while amongst them coursed the smaller arteries and veins, running often for a long distance parallel to each other, and in the direction of the axis of the bone, without altering their calibre much before they broke up into dense bouquets of capillaries. In no case whatever did we see any lymphatic enter this layer, although, as shown in fig. 2, they were very plentiful on the outer surface and within the fibrous or gelatinous tissue which composes that surface, but they were not found at all on the inner, cell-studded stratum which exists so plentifully in young animals. To this stratum Ranvier and some other histologists have applied the grotesque name of subperiosteal marrow, a term quite unnecessary, as there is really no difference in character, although there is in quantity, between the cells found there and those found all through and on the outer surface of the periosteum. It is obvious that it is the nuclei belonging to these cells, that are found in such numbers on the periosteum of young animals, which has justified Professor Robin's interpretation of the periosteum as being a cellular tissue. The fact, however, is that the base or matrix of the membrane is gelatinous, and the cells embedded in it are of varying quantity, varying according to the age or species of the animal, or the locality of the bone. It is unfortunate that hitherto all investigation into the structure of the periosteum, or into the relations of its component elements to each other, seems to have been made only by means of transverse and longitudinal sections through the thickness of the membrane. Such sections, when stained in the usual banal fashion, with colouring reagents like carmine or hæmatoxyline, give as correct an idea of the general arrangements of the elements therein as sections through the canvas of an oil painting would give a conception of the picture

upon its surface. When the cells, vessels, and nerves contained in the periosteum have had their outlines clearly mapped out with silver solution, and they are studied in their various superficial and deep planes, another conception is acquired of their relation to each other than that given in the text-books, which has been derived from transverse sections only. It is this consideration which has caused us to define, a little more correctly than is generally done, the component structure of the periosteum the better to enable our description of its lymphatics to be understood.

What we have just said may render more intelligible our explanation of the different aspects presented respectively by the lymphatics in fig. 2, from the periosteum of a young goat, and in fig. 1, from that of an old horse. The periosteum in fig. 2 was comparatively thick, and the lymphatics seen there are principally efferent vessels. On either side of the main line of vessels branches are given off, which seem to end abruptly. These abrupt terminations, however, are not the terminations of the lymphatics, but only mark the points where they left the outer surface to ramify more deeply within the membrane, and to join there with each other, and with similar lateral branches from similar main groups of efferent lymphatics. An idea of those deeper intercommunicating lymphatics is given in fig. 4, from the deeper structure of the thick periosteum of a young man, of which fig. 5 shows a fragment, of a superficial lymphatic, lying upon a bed of fat cells on the outer surface of the membrane. In figs. 1, 3, and 5, on the other hand, from an old horse, we have the periosteum of minimum thickness, so thin, indeed, that, both outer and inner surfaces having been stained with silver, it was possible to see the component elements on either surface and in the thickness of the membrane, without changing the focus of the microscope, under a power of thirty diameters. The consequence of this is that the whole of the lymphatics of the membrane lie completely and distinctly upon or above the outer surface, and we have thus presented to us, in one plane, the combined collecting and efferent lymphatics of a whole tissue or region. In our paper on the lymphatics of the urinary bladder we described, for the first time, an arrangement of lymphatics wherein the collecting

and efferent vessels were combined in the one group, and applied apparently to the drainage not of a surface or of a solid tissue, but specially of groups of large blood-vessels, in such a way that the fluid portion of the blood, which under the normal pressure must exude to a certain extent through the arterial walls, would at once pass into the lymphatics before it had an opportunity of soaking into the neighbouring tissues, and thus be immediately carried off from the organ through the lymphatics. In the bladder, however, those lymphatics, as shown in figs 2 and 6, were exceedingly irregular in size and arrangement, winding tortuously around and between the blood-vessels. In the periosteum, on the contrary, as shown in fig. 1, we have the very beau ideal of a regular arrangement of lymphatics, combining in each vessel collecting and efferent functions, and applied solely to the drainage not of a surface but of lines of blood-vessels. At page 369 of the article referred to, we gave merely suggestively the hypothesis of a combined collecting and efferent function to those lymphatics, as being adapted specially to the lines of large blood-vessels, but in the periosteum the matter is placed beyond doubt. There the lymphatics are applied to the large blood-vessels above, not only with scientific drainage adaptation, but with mechanical exactitude and regularity. On either side of each group we have a large lymphatic running parallel to the group, and providing that any of the fluid portion of the blood which may exude through the walls shall not escape laterally, while, as if to obviate the possibility of any fluid passing off from the surface, we have two smaller lymphatic drains running parallel to the central artery of the belt. That, in such a specimen of periosteum, the lymphatics are applied solely to the lines of blood-vessels, and not to the surface or component tissue of the membrane, is placed beyond doubt by observing that the great mass of the surface, in what may be called the centres, *m*, *m*, of the vascular meshes, possesses no lymphatics whatever, being protected by the outlying parallel lymphatics, while the fact that the plexus of lymphatics is complete, and sends no lateral erratic offshoots off into the substance of the periosteum, as seen in fig. 2, shows that the interior or body of the membrane has no lymphatics.

Attached to the ends or junction of the straight and parallel

lymph drains, we find in many cases dilated portions or ampullæ, which act, no doubt, as temporary reservoirs as occasion requires, while as, even in combined collecting and efferent lymphatics forming meshes, there must be some objective points on the surface to which the flow of collected lymph is determined, so do we find here and there on the surface arrangements like that seen in fig. 3, also from an old horse, these being the points from which what may be called the extrinsic efferent lymphatics receive and carry off from the periosteum the lymph which has been collected upon it. In no case, whatever, even on the largest bones of such large mammals as man and the horse, have we found any muscular or other strengthening elements upon the intrinsic lymphatics of the periosteum. Even upon such comparatively large sacs or dilatations as are seen at fig. 6, the walls were composed only of the one layer of crenated endothelium.

It may be asked if there is a difference in the appearance or arrangement of the lymphatics on the same surface in young and old animals of the same species, how or at what period is the change effected? Our answer to this is, that we believe that the lymph vascular system, like the blood vascular system, as we have shown elsewhere,<sup>1</sup> may be continually changing its position in the tissue, according to the necessities of the time or place. The fine blood-capillaries and the large intrinsic lymphatics which correspond to the capillaries in the structure of their walls, may be formed here to-day by the skilful auto-adaptation of the wandering or embryonic cells which came for that purpose, and to-morrow the same vessels may become occluded if no longer necessary, and their component cells break up their connection, and travel away elsewhere as mysteriously as they made their appearance there. This probably explains the difference in the appearance in the lymphatics of thick and thin periosteum of young or old animals, for there is no reason why the lymphatics should not be modified by the same causes which modify the number and position of the cells and capillaries in the same locality, the few cells and capillaries, for example, seen in fig. 6, being a mere fraction of the immense

<sup>1</sup> "On the Development and Retrogression of Blood-Vessels," in *Journal of Royal Microscopical Society*, 1880, fig. 13, plate xv.

masses which existed there when the animal was young. This immense reduction in the available supply of blood-vessels, lymphatics, and branched cells, is probably the chief factor in the diminished nutrition of the bones of the aged that leads to their brittleness, and above all to the retardation of the healing of fracture. That, however, is too large a question in surgical pathology for us to do more than indicate the chief factor here.

The presence of the so-called branched cells of the connective tissue in such varying quantities in the periosteum reopens an important question, which we have referred to more than once in these pages and elsewhere, but which we are now better able to settle than at any time previously. We have also discussed the possibility of their being the radicles of the lymphatics, in accordance with the hypotheses published long ago by both Virchow and Von Recklinghausen. We have rejected these hypotheses absolutely, and shown that in the great majority of tissues in which such cells were found, they were generally conspicuously absent from the neighbourhood of the lymphatics, and unconnected with them, although plentiful in the neighbourhood of the blood-vessels. In our paper on the lymphatics of the pancreas, we even demonstrated the cause of the artificial and erroneous appearances which had led many to suppose that these branched cells were the radicles of the lymphatic system. We, however, admitted that although that hypothesis, or indeed any hypothesis which conceived a system of lymph radicles lined with protoplasmic elements, was wholly wrong, yet in the future it was possible that a system of fine channels might be demonstrated without any protoplasmic lining, and which, therefore, silver imbibition would be unable to show. We no longer make this or any other reservation, and deny absolutely the existence of lymph channels other or smaller than those shown in drawings accompanying our various publications. The proof of what we state is easy, and is contained in the method of injecting dilute silver solutions into the lymphatics which we have described at the commencement of this paper.

Anyone who has injected the blood vascular system with dilute silver solution is aware that the fine blood-capillaries are made abundantly evident by it, and they can conceive that if any channel as small existed in connection with the lymphatic



system, these small channels would be easily demonstrated. Now, this is what we have put to the proof, not only in the periosteum, but in other tissues where great valveless collecting lymphatics exist. Having succeeded in getting the hypodermic needle to open into such lymphatics, and seen the silver solution pass along the lymphatics, we have, as already described, closed the outlet with our finger nail, so as to cause considerable pressure within the plexus, even to bursting of a vessel. When such tissues are subsequently prepared for microscopical investigation, we see only the distinctly marked intercellular outlines of the endothelium forming the lymphatic wall, and no small openings or passages in, at, or from it. Had there been any cell-lined radicle passing from the lymphatic, it would have been shown, as well as the outlines of its lining protoplasmic elements. Had there been naked passages without any such lining, they would have been shown even more prominently, if their walls were unprotected from the action of the silver; they would have stained as black as silver albuminate generally appears when exposed to light. But there was never anything of the kind, so that the whole tottering fabric of lymph radicle hypotheses may be allowed to fall once and for ever. We ought, however, to state here that, in admitting as we did (*vide* page 373, vol. xv.) the possibility of the existence of a minute system of lymph radicles not lined by protoplasmic elements, which would otherwise have been shown by silver, we were led to do so in consequence of the drawings given by that veteran investigator, Professor Sappey, in several of his recent works, and more especially in the large folio plates illustrating his great work on "Lymphatic Vessels," now in course of publication. He there depicts close and intricate plexuses of very fine channels as the radicles of the lymphatics; and as these have dilated portions at intervals, he has applied to them the names of lymphatic "capillicules" and "lacunes." The distinctness with which these were drawn made it impossible for us to doubt their existence; hence our qualified admission. Since then, however, by the kindness of Professor Sappey, one of us has had the opportunity of examining the original preparations, and of seeing similar preparations made by him in our presence. On carefully examining those preparations, we were there and then

enabled to state unhesitatingly that the so-called lymphatic radicles were only the blood-capillaries of the various regions, distorted almost beyond recognition by the use of powerful reagents. This explanation may be of service to other investigators, who may have felt some doubts as to the nature of the lymphatic radicles described and figured by Professor Sappey.

For pure physicists the question has an interest apart from that of the lymphatic system, in connection with the dialysis of liquids. If lymph or fluid can get naturally and easily into the lymphatics, it ought equally naturally and easily to get out again when internal pressure is applied. But by what channels? By no channels. In cases where we have made prolonged injections of the lymphatics, and given them excessive exposure to light afterwards, a certain amount of territory around and external to the lymphatic wall shows that the silver influence has travelled outwards. The staining of the circumambient tissue is, however, that of a dark, soft haze, in which the highest powers of the microscope show no differentiation of tissue into channels or tissue elements; but when the injection has been used forcibly to burst the vessel and cause dissociation of the tissues, a very different aspect is presented. The same confused aspect is seen in all the numerous cases in which the injection has failed to find or run into the lymphatics, but has caused dissociation of the tissues instead. With silver solution at our disposal for injection, it is mere bungling to use Berlin blue or asphalt injection for the *research* of the lymphatic system. Where the silver passes it marks its path, showing the lymphatic endothelium of the wall when successful, and the débris of albuminates when a failure; but the coloured fluids referred to only show themselves, and give no indication whether they are in a lymphatic or forming an extravasation, unless when in an efferent lymphatic distinct valvular dilatations are shown.

Apart from Dr. Budge's one figure of the supposed lymphatics of the periosteum, we have spoken of his *schematische* drawings of what he believed were the relations of the lymphatic radicles to the cells of the bone, which he portrays as being surrounded or bathed by the lymph. With this are to be coupled the statements of those who speak of having injected lymphatics coming

out of bone, and of having been able to trace the vessels backwards into the cancellous tissue of the bones. Now, all our successful injection of the lymphatics of the periosteum have shown us (what, indeed, any little knowledge or consideration of the anatomy of these vessels might have led anyone to expect) that it is impossible to inject these vessels backwards, and that in every case the injection fluid, when once it got fairly within the lymphatics, took the shortest path down the lymphatic stream, never upwards; and even when, as we have described, we closed the outlet, and pressure was brought to bear on the interior of the vessels, it merely closed firmly the valves of the various branches leading into the main stream, and thus effectually prevented all regurgitation backwards, the silver solution in all cases making the distended form and endothelium of the valvular dilatation and their valves distinctly visible.

This result, and the very consideration of the anatomy of the vessels, merely accentuates Professor Sappey's condemnation of the statements referred to as being without any value. In regard to Dr. Budge's theory, in particular, we may observe that to obtain the appearances which he considers to warrant his hypothesis, that is, to drive a coloured fluid into the substance of the bone under great pressure, is a mere physical operation, and has nothing whatever to do with the presence or absence of lymphatics in that tissue.

In addition, however, to the mere consideration of the arguments which prove the unfounded character of the statements referred to, we have endeavoured, when injection failed, to obtain some results from silver imbibition that might bear on the question of the existence of lymphatics on the surfaces of bone unprovided with periosteum. Choosing a bone whose marrow cavity was large, and the wall free from spicules on the surface (and for this purpose we found the humerus of the horse the most convenient of any), a portion of about two inches in length was first sawn off, and carefully split through the centre with a sharp cleaver. In this way each half of the cylinder of marrow could almost be shaken out, so destitute was it of any adhesion to the bone cavity. A solution of silver was then applied to both the surfaces of the marrow and of the wall of the cavity of the bone. The latter was then placed for a couple of days in a

dilute acid solution, which dissolved the earthy salts sufficiently to allow the surface to be sliced off in thin sections. When such sections were examined under the microscope, the spaces occupied by the bone cells were quite distinct, but nowhere was there any trace whatsoever of lymphatics. The smooth, free surface of the marrow which had been left quite uninjured, and treated with silver, gave the same negative result. The outlines of the fat cells of the marrow were made very distinct, as well as those of a few branched cells, and of the smaller blood-vessels supplying the marrow, but there was no evidence of the presence of any lymphatics on that surface. This search we repeated several times on different bones, so that a comparatively very large extent of marrow surface and surface of the bone cavities was carefully examined by us without our finding any trace of the presence of lymphatics. And it is this which leads us, apart from the considerations we have mentioned, to reject the conception of the presence of lymphatics of the bone itself, apart, that is to say, from those of the periosteum, the outer surface of which appears to be the true lymphatic periphery of the whole osseous apparatus.

When a careful survey is made of the relations of the lymphatics to the blood-vessels on the outer surface of the periosteum, it is found that the larger lymphatics, almost without exception, lie superficially in regard to the blood-vessels, as if they had been applied to that surface after the others had been placed there, and one feels that if an animal large enough could be found whose lymphatics bore any comparison to its size, it might be possible to dissect off the lymphatic plexus without disturbing the blood-vessels. The connection between the two also seems to be of the loosest kind, as shown when fat cells develop on that surface.

In young animals it is seen that, on the outer surface of the periosteum, a large number of branched cells exist, lying alongside of the blood-capillary plexuses also seen on that surface. It is from those branched cells, probably wandering cells, that the fat cells which exist only on that outer surface are developed. When this development of fat tissue takes place, it seems to have the effect of raising the lymphatics up from the surface of the blood-vessels, the fat tissue being in a manner interposed

between the two, as shown in fig 5. It is generally difficult to show the lymphatics under such conditions, in the first place, because in dissecting off the loose tissue from the surface, before applying the silver, the fat gets shed, with the result of making the surface so to speak waterproof, and impervious to the silver solution, which therefore fails often to give the ordinary reaction; in the second place, the silver may have reacted successfully, and the lymphatics may escape notice, from the fact that the fat cells, being protoplasmic on the surface, form a white ground substance to the white lymphatic. Using, however, a high magnifying power, and looking out especially for the characteristic outline of the endothelium cells which form the lymphatic wall, enable us to recognise the whole lymphatic plexus, lying superficial to the layer of fat tissue which intervenes between it and the gelatinous tissue or matrix, with its blood-vessels and nerves, that form the outer portion of the periosteum.

It must not, however, be imagined that there are lymphatics wherever there is periosteum. In the dura mater of the skull in the child, which is regarded as periosteum, there exists what at first sight appears to be a dense irregular plexus of lymphatics. Apart, however, from the peculiar shape of the endothelium forming the walls of these vessels, careful examination shows them breaking up into fine branches, getting smaller and smaller as they divide and subdivide, until they become of the size of blood-capillaries. They are indeed blood-capillaries, and the large vessels forming the plexus in question are only veins, and not lymphatics, for lymphatics never break up into branches in that fashion, numerous drawings in numerous published researches into the lymphatics notwithstanding, which by that appearance alone may be recognised as erroneous, as we have already described and instanced in this *Journal*. The peculiar arrangement of the venous system on the dura mater of the skull is no doubt a special adaptation to the part, like the wonderful venous plexuses on the surface of the spinal canal, to act as a buffer for obviating the effects of jars and shocks on the delicate nerve structures within them. We may also add, when on this subject, that we have carefully prepared with silver, and examined under the microscope, the internal surfaces of the thin skulls of scores of small mammals, and have never yet been able to

detect any vestige of a lymphatic upon them, although they were to be seen on the outer surface of the skull in the periosteum which covers that surface.

While the chief function of the lymphatics on the peripheral surface of the osseous organ is that of a drainage system, we ought also to bear in mind the part it may take in the process of repair in the case of fracture of the bone. This, however, pertains more to the question of the formation of callus, a subject upon which we shall not enter further at present, although it would scarcely be any exaggeration to say that hundreds of authors, both surgeons, pathologists, and histologists, have written about it without knowing whence the original material which formed the callus was derived. They have, indeed, said it came from the periosteum, but as hitherto all the attempts of investigators to discover lymphatics in the periosteum have been unsuccessful, it was a difficult matter to explain where the so-called plastic lymph came from to form the callus. Connected with this, however, and with the remarks which we made in our last paper regarding the flow of lymph that succeeded to the stoppage of the bleeding from a cut in the skin, there is an observation upon the character of the structure of the walls of lymphatics which we have to offer. In nearly every paper which we have published describing the lymphatics of various organs or tissues, we have stated, in opposition to almost every writer on the lymphatic system, that in these tissues or organs the walls of the intrinsic lymphatics never possessed any muscular or contractile elements, being formed, however large their dimensions, by the one layer of thin, crenated, endothelial cells joined edge to edge. This is stated as a mere anatomical fact, and as opposed to the statements that the walls of the lymphatics consisted of several layers, but we saw no physiological importance in the fact which we so continually reiterated. It now appears to us that for the purpose of repair there is a definite plan in the non-contractile character of the wall (apart from the endothelium itself), which accounts for the lymph continuing to flow after the bleeding has ceased, in order that the lymph may take the place of the blood at the spot where repair is to be effected, as seen in the case of a wound of the skin. So in the case of fracture of a

bone and rupture of the vessels of the periosteum, the arteries may be expected to close immediately after the first rupture, and with the effusion of only a small quantity of blood from the capillaries and veins of the part. But following this, and continuing long afterwards, we have a flow of reparative material from the great lymph channels of the part, which flow could not have taken place had their walls, as in the case of the great extrinsic lymphatics or lacteals, been provided with muscular contractile elements. The "plastic lymph" seems to take the place of the blood around the broken extremities, and within it the embryonic cells organise the tissue that repairs the fractured bone.

On the action of the lymphatics as the so-called absorbents in causing absorption of bone, we need add little to what we have already remarked upon John Hunter's vagaries regarding this question. The complete answer to his typical examples is that the lymphatics are as far removed as possible from the very surface where absorption is going on, and as they do not exist on or near to the surface which is being absorbed, they cannot well be the agents in performing the absorption. Some modern surgeons, indeed, misled by their experiments, which have so often given contrary results, deny altogether that bone is ever absorbed. Of these Professor Spence is a notable example, for in the posthumous edition of his work on surgery he states that "no one now-a-days believes in the absorption of bone." Yet the examples quoted by Hunter of such absorption are as plentiful at the present day as they were in his time, as seen in the absorption of the alveoli of the jaw from which the teeth have been removed, of the sternum or vertebral column through pressure of an aneurism, and even physiologically, as seen in the normal enlargement of the cavities of the long bones during the period of growth. Nature's examples of absorption of bone being more satisfactory than the contrary results obtained from experiments on animals, the only question which remains is to find out the agents in the process. That the lymphatics cannot so act we have shown abundantly, and the only other available agents it seems to us are the wandering or embryonic cells, which lie on the under or bony surface of the periosteum, on the exterior of the bones, and which exist in such numbers

in the marrow cavities that at the present day these cavities are looked upon as the great breeding places for both white cells and red corpuscles. These cells are therefore in our opinion the only agents which deserve the name of the absorbents of bone.

Finally, the anatomical conditions which we have found to exist in the lymphatics of the periosteum may be shortly expressed in the following conclusions:—

1. The lymphatics of the periosteum exist only on the outer surface, or within the outer gelatinous (white fibrous) stratum of the membrane. They never reach, or ramify upon, the inner or bone surface of the periosteum, and this holds good for all animals and all ages that we have as yet examined.

2. When the periosteum is thin, more especially when the animal is old, the whole lymphatic plexus lies free upon the outer surface. When the periosteum is thick, and a deep cellular stratum exists on the inner surface, as in growing animals, the smaller twigs pass beneath the outer surface, but never reach or enter the cellular stratum.

3. In the great majority of cases, the lymphatics seem to be specially and regularly arranged for the drainage of the main lines of blood-vessels, which ramify like a network on the outer surface of the periosteum, where they are accompanied by the lymphatics. The lymphatics seldom enter the area of the large open meshes thus formed, or leave the companionship of the large blood-vessels.

4. No lymphatics exist on the surface of the great cavities in bone, where the marrow lies directly in contact with the bone. There is thus every reason to believe that the lymphatics never come in contact with the bone itself, and that bone possesses no lymphatics apart from those found within the periosteum, which may be physiologically considered, therefore, as the lymphatics of bone.

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#### DESCRIPTION OF PLATE XIV.

*(Drawings made by the aid of the Camera Lucida, and afterwards reduced by Photography.)*

Fig. 1. View, under a low power, of the whole of the lymphatics of a portion of the periosteum, from the tibia of an old horse. These



lymphatics are specially adapted for the drainage of the main lines of blood-vessels; they lie wholly on the outer surface of the periosteum ( $\frac{1}{30}$ ).

Fig. 2. Similar view of lymphatics, chiefly efferent, from the outer surface of the periosteum of the tibia of a young goat. A little to the right of the centre of the figure is neutral ground, where the terminal twigs of two great lymphatic streams, flowing in opposite directions, meet. The lateral afferent branches, *al*, lie immediately beneath the outer surface, but never reach the inner surface or cellular portion of the periosteum ( $\frac{1}{30}$ ).

Fig. 3. Similar view of the whole of the lymphatics on the outer surface of a portion of the periosteum, from the humerus of a horse. This figure represents one of the centres towards which the various plexuses, similar to those seen in fig. 1, direct their lymph streams, preparatory to the lymph passing away from the periosteum, which it did a little beyond the centre of the upper edge of the figure ( $\frac{1}{30}$ ).

Fig. 4. Shows very fine lymphatic twigs connecting the larger and more superficial trunks, from the deeper stratum of the outer or gelatinous portion (white fibrous) of the periosteum of the tibia of a young man ( $\frac{1}{75}$ ).

Fig. 5. Shows a portion of one of the large lymphatics, from the same preparation and under the same magnifying power as fig. 4. It lies on a bed of fat cells, on the free outer surface of the periosteum ( $\frac{1}{75}$ ).

Fig. 6. Shows the difference between *ll*, the lymphatics on the outer surface, *cc*, the blood-capillaries, and *bb*, the branched cells on the inner or bone surface of the periosteum. This is only a more highly magnified portion of the preparation drawn in fig. 3, taken from a little above the centre of the figure. These blood-capillaries will, on examination, be seen to be identical with what Budge supposed to be lymphatics. To prevent undue complexity, only a small portion of the ground substance, with its branched cells of the inner surface, has been inserted at the upper right hand corner of the drawing. In this case the periosteum was so thin that, after drawing the lymphatics on the one surface, the structures on the other surface were next drawn, without moving the preparation, and with very slight lowering of the focus of the microscope ( $\frac{1}{75}$ ).

Arteries, *a*; veins, *v*; lymphatics, *l*; fat cells, *f*; branched cells, *b*; nerve bundle, *n*; meshes, *m*; blood-capillaries, *c*.

THE BRACHIAL PLEXUS OF THE MACAQUE MONKEY  
AND ITS ANALOGY WITH THAT OF MAN.<sup>1</sup> By  
W. TYRRELL BROOKS, (*late Demonstrator of Anatomy*),  
*Demonstrator of Physiology in King's College, London.*

FROM a comparison of six dissections which I have recently made, I find that the brachial plexus of the Macaque Monkey includes in its origin the whole of the 5th, 6th, 7th, and 8th cervical nerves, while it receives from above a considerable branch of the 4th cervical, and from below the main trunk of the 1st dorsal nerve. The 4th cervical, which is a nerve of considerable size, gives off a branch, which, running downwards, joins the small 5th cervical just as the latter gives off the phrenic nerve.

This is constant, and some of the fibres from the 4th cervical may be traced passing over the 5th (though apparently blended with this nerve), and entering the phrenic.

The 5th cervical, after having received its communicating branch from the 4th, runs to join with the 6th cervical, forming with this nerve an upper trunk, from the commencement of which springs a large nerve, the suprascapular. This is an arrangement entirely constant, and agrees with the origin of the same nerve in men, with the slight exception that it takes more of the fibres of the 6th than in the similar human nerve. After this the upper trunk divides, giving off one branch running anteriorly to the other.

The anterior branch is longer than the upper trunk itself, and unites with a branch from the 7th cervical, forming by its union an upper cord, which is short.

From the anterior surface of this upper cord springs the external anterior thoracic, whilst the cord itself, after giving off the external cutaneous nerve, ends as a large branch, which, running downwards, forms the upper or outer head of the median.

<sup>1</sup> In referring to the human plexus, I have used chiefly Quain's *Anatomy*, vol. i. (last edition); Mr. Clement Lucas's paper on the "Arrangement of the Human Brachial Plexus" (*Guy's Hospital Reports*); and Dr. J. Henle's *Anatomie Des Menschen*.

The posterior branch from the upper trunk is larger than the corresponding anterior branch, and descends to meet a similar branch from the 7th cervical, giving off in its course two small subscapular nerves (this being an arrangement frequently met with in man), whilst from its point of union arises a considerable trunk, which, after running outwards for a distance, subdivides into the middle subscapular and a large nerve the circumflex. After originating this branch, the conjoined posterior branches of the upper trunk and 7th cervical pass downwards, and, uniting with a posterior branch of almost similar length arising from the combined 8th cervical and 1st dorsal, form a very large nerve, the musculo-spiral.

Besides this posterior branch of the conjoined 8th cervical and 1st dorsal trunk, another cord, lower in position and longer, arises. This gives off from its face the internal anterior thoracic, and continuing on subdivides into two nerves of almost equal calibre, the upper one of which unites with the branch from the outer cord to form the median nerve; the lower continues a straight course (giving off from its lower side, close to its point of origin, the internal cutaneous nerve), and is the ulnar nerve.

These are the chief of the regular nerves of the brachial plexus originating below the clavicle. I will now go on to describe some of the less regular branches, together with those that arise above the clavicle. A very constant nerve is that to the subclavius muscle; it arises from the 6th cervical, and gives, besides its muscular branch, a considerable branch, which, running over the scalenus anticus, unites with the phrenic as this is disappearing into the thorax.

This branch is a very constant one, and in the only case in which I have failed to find it, the phrenic obtained fibres from the 6th cervical by taking one of its roots from this nerve directly.

The phrenic arises from the 5th cervical, just where that nerve receives the communicating branch from the 4th, and has a communicating branch of large size from the nerve to the subclavius.

Its two varieties are, either that it has separate roots from both 4th and 5th cervical, or, as I mentioned above, where it fails to receive fibres from the 6th through the nerve to the subclavius, it may obtain a special root from the 6th itself: it is

constant, therefore, in this respect, viz., that it obtains in some way or other nerve fibres from the 4th, 5th, and 6th cervical nerves. Coming off from the back of the plexus, and most constant in its origin, is the nerve of Bell. This important nerve arises by two heads from the 6th and 7th cervical nerves respectively shortly after they emerge from the intervertebral foramina.

The nerve to the rhomboids seems a little uncertain in its origin, but most generally it comes from that head of the nerve of Bell which arises from the 6th cervical. It may, however, have a higher origin from the 5th cervical nerve direct. (Lucas, in his description of the human brachial plexus, mentions the origin of this nerve from the upper head of the posterior thoracic nerve as a regular arrangement.)

The long subscapular is much less constant in its origin than its fellow of the same name, in one case arising from the posterior branch of the 7th nerve as this was going to form the upper head of the musculo-spiral, in another case from the back of the musculo-spiral trunk itself, whilst in a third it arose still nearer the bottom of the plexus by coming from the lower head of the musculo-spiral (*i.e.*, that head which arises from the conjoined 8th cervical and 1st dorsal). In a fourth case it took its origin from the posterior branches of the 7th cervical and of the conjoined 8th cervical and 1st dorsal (coming in fact from the two heads of the musculo-spiral). Its origin, therefore seems constant in always being related to the musculo-spiral in some way.

The internal anterior thoracic always receives a branch from the outer cord, in some cases coming from this directly, in others arising as a branch from the external anterior thoracic, entering the internal thoracic before this supplies any muscular twigs.

The lesser internal cutaneous nerve was in some cases absent, its place being supplied by a large branch from the intercostal of the 2nd dorsal; in one case it arose from the internal cutaneous, and in another from the internal anterior thoracic.

The nerves to the scaleni and longus colli are uncertain in their origin, coming irregularly from the upper nervous cords. The similarity of the above description with the brachial plexus in man is evident. The chief peculiarities are the following:—

The 4th nerve is larger, and its branch to the 5th of more

importance, the 5th itself being considerably smaller than in man.

The 7th nerve divides into its anterior and posterior branches almost immediately on leaving the intervertebral foramen, and not, as in the human plexus, remaining undivided for one or two inches.

The 8th and 1st dorsal unite much earlier, and in the Macaque Monkey form a large thick cord, which, until traced close to its origin, appears to be only one nerve.

The 1st dorsal nerve is smaller comparatively than is usually found in man. (Henle, however, in his drawings, makes the 1st dorsal in man to bear very much the same relation to the 8th cervical as that which I have found in this series of dissections.)

The phrenic, which, in man, is usually described as arising mainly from the 4th cervical, and having its accessory roots from the 3rd and 5th, here arises somewhat lower, receiving a constant origin from the 4th and 5th, and generally obtaining its fibres from the 6th cervical through the nerve to the subclavius, sometimes, however, having a direct root from the 6th itself. I have not found any communicating branch from the 5th and 6th nerves to the phrenic just as this enters the thorax, which is a frequent arrangement in man.

The nerve to the subclavius is a comparatively large one, and arises solely from the 6th, and not as in man from the 5th and 6th. (In one of Henle's plates of the brachial plexus he figures this nerve as coming off direct from the 6th.)

The external respiratory has a lower origin than the same human nerve coming from the 6th and 7th instead of from the 5th and 6th. The origin of the circumflex and middle subscapular from the posterior branches of the conjoined 5th and 7th, and of the 7th, is dissimilar to man where the circumflex originating from the posterior cord receives fibres not only from the 5th, 6th, and 7th, but from the 8th cervical and 1st dorsal also.

The external anterior thoracic arose always in my dissections, by one head, and not, as is usually described in the human body, by two. There was a very constant branch from this nerve to the internal anterior thoracic. This, however, frequently occurs in man, and is figured more than once in Henle's plates.

## A CASE OF PRIMARY SARCOMA OF THE PLEURA.

By R. W. GREENISH, F.R.C.S. (PLATE XV.)

PRIMARY sarcoma of the pleura has been so seldom met with, that the following case deserves, I think, being put upon record, especially as its origin presents several points of interest.

The patient, aged 54, was admitted into hospital with what appeared to be simple chronic bronchitis; she was thin and pale, lips and finger nails somewhat blue, troublesome cough, and very short of breath. Physical examination revealed only a few râles over chest. She improved so much under treatment as to be able to leave her bed. A month after admission she became much worse; breathing was very rapid, and there were all the signs of obstruction to the circulation, such as blueness of lips, &c. Physical examination showed, in addition to the above râles, dulness at the left base. Breathing became steadily worse, and she died in the course of twenty-four hours. All the history that could be obtained was that her first husband and one child died of phthisis; no history of tumour of any kind.

*Post-mortem.*—Twelve hours after death. Heart right side distended with blood. Right lung congested; on pleura a small cicatrix. Left side of chest full of fluid. Pleura, pulmonary, costal and diaphragmatic much congested, and covered more or less thickly with small nodules; these were most abundant over upper part of costal, and on diaphragmatic pleura, but attaining their largest size on the former; over the rest of costal pleura and on lung they were much less numerous. The majority of the nodules varied in size from some only just visible, to that of a small pea; they were irregularly rounded in shape, and often markedly nodular; some were sessile, others attached by a pedicle, which in some cases was extremely thin; all those of large and medium size showed patches of pigment on the surface, and round the more sessile growths was a zone of congested vessels. On section they were hard and cartilaginous, had a greyish semitranslucent appearance, generally with patches of pigment, and often containing an opaque white patch in

centre. Some of the nodules terminated on their free end in a thin fibrous cord, varying from 1 to 3 and 4 centimetres in length and occasionally two or three were seen matted together by such tissue. One nodule on the upper part of the costal pleura differed from the others as regards its size; it was a large irregular sessile growth 2 centimetres long by 1 broad and  $\frac{1}{2}$  in thickness. In the diaphragmatic pleura the nodules were all small, but so numerous that the serous membrane was in great part converted into an opaque membrane from 1 to 2 millimetres in thickness. They appeared at first as small flat elevations, situated just under the serous membrane, which they gradually raised, while the seat of attachment became more and more constricted, finally resulting in a freely movable pedunculated nodule. The condition of the pulmonary pleura was a little different, the growth was level with the surface, occasionally in small circular patches, but oftener having a radiated arrangement. On section they were wedge-shaped, the point of the wedge being directed inwards. There was little to note in the other organs; the right lung was congested, lymphatic glands slightly enlarged. Kidneys were congested, left contained a small abscess just underneath the capsule. Liver, spleen, and intestines healthy; ovaries studded with small cysts. In the cervical canal of the uterus were a few patches of superficial ulceration.

*Microscopical Examination.*—When examined under a low power all the nodules showed in the main the same structure, viz., a more or less developed alveolar arrangement, the alveoli being filled with large epithelioid cells, as in fig. 1, which shows the section of a nodule taken from the costal pleura; the fibrous tissue was in all abundant, in some very abundant, but what was more striking was the extreme irregularity in the shape and size of the alveoli, some being large and rounded, others small, and appearing as mere fissures in the fibrous tissue as at *a*. Even under a low power fine bands could be seen passing across the spaces, as at *b*; but under a high power this apparent alveolar structure almost entirely disappeared, bands of fibrous tissue being seen dividing the alveoli into small spaces mostly holding three or four cells, occasionally more. By pencilling as in fig. 2, this structure came out extremely

clearly. The cells were large, containing a well-marked nucleus and often nucleolus; they closely resembled those of cancer, but differed in being subject to more variety in size; the protoplasm was often very ill-defined, and both it and the nucleus stained but very imperfectly, hence rendering it frequently a matter of difficulty to distinguish the body of the cell from the adjacent fibrous tissue. Towards the circumference of the nodule the connective tissue became gradually denser, and formed a sort of capsule, as at *c* (fig. 1), which here and there contained blood-vessels. On its deep surface was a layer of fibrous tissue, the thickened subserous structures, to the contraction of which the pedunculated condition of many of the nodules was due. In none of the larger nodules could any blood-vessels be seen, only in the capsule.

Those on the diaphragm differed in containing more fibrous tissue, and in the alveolar spaces being here and there very much larger, containing as many as twenty or thirty cells, without any bands subdividing them. In many sections the serous membrane between the nodules was infiltrated with these cells, some occurring singly, others in rows, others again in small groups, while the fibrous tissue was increased in quantity, and both it and the subserous tissue, but especially the latter, infiltrated with round cells. As regards the lymphatic vessels it was very difficult to speak with certainty; the larger vessels, especially those of the subserous tissue, were nearly always free; very seldom could I see anything that resembled a lymphatic vessel filled with cells, even in sections taken parallel to the surface as recommended by Schulz (*Archiv. der Heilkunde*, B. xvii.), and this was always when the process was moderately advanced, never in the early stages, and chiefly in those from the diaphragm. The nodules from the lung differed from either of the above. Here two layers could be made out—a superficial one, in which the connective tissue was extremely well developed, and very rich in cells, the alveoli being often widely separated from one another; these too were much larger than in the parietal pleura, often had a direction parallel to the surface, and were occasionally provided with processes, which anastomosed with those from neighbouring alveoli. Blood-vessels were also very frequently met with in this layer. In the deeper



stratum the epithelioid cells increased in number, while the fibrous tissue became less and less, till little but cells were to be seen. The adjacent lung alveoli were generally filled with epithelial cells, and their walls somewhat thickened; and thus they came to closely resemble the structure of a nodule, differing only in the size of the alveoli.

*Formation of the Nodules.*—The first change to be noticed was that the serous membrane became thickened at one point, and the subserous tissue infiltrated with round cells, which were mostly situated round the blood-vessels. The thickening of the serous membrane was due partly to an increase of the fibrous tissue, partly to its being infiltrated with round cells; these latter occurred either singly, or more often in rows of three, four, or more, as at *a* (fig. 3), which is taken from the edge of a small nodule on the parietal pleura; the cells appeared to remain only a very short time in this condition, but gradually acquired a zone of protoplasm round them (*b*, fig. 3), which increased till they assumed the appearance of large epithelial cells, as at *c* and *d*, sometimes being in groups, *c*, sometimes in long rows, *d*. These cells were thus evidently derived from the ordinary connective tissue cells.

Several cases of the same form of sarcoma of the pleura have been described by various authors. Thus, Lebert<sup>1</sup> gives details of three cases. Bidal,<sup>2</sup> Lepine,<sup>3</sup> Böhme,<sup>4</sup> Schulz,<sup>5</sup> Birch-Hirschfield,<sup>6</sup> Eppinger,<sup>7</sup> and Unverricht<sup>8</sup> have each observed a case, which with mine make eleven in all. These cases all resembled one another in that the disease occurred as an infiltration of the pleura, converting it into a thick layer of hard cartilaginous tissue from one to several centimetres in thickness. In two cases was the disease confined to the right, in six to the left, and in three were both sides affected, though in one case, Schulz's, the disease was more advanced on the left side; thus really making

<sup>1</sup> *Traité d'anatomique pathol.*

<sup>2</sup> *Bulletins de la société anatomique*, tome xxvii.

<sup>3</sup> *Ibid*, tome xiv.

<sup>4</sup> Virchow's *Archiv*, Band 81.

<sup>5</sup> *Archiv. der Heilkunde*, B. xv.

<sup>6</sup> *Lehrbuch der pathologischen Anatomie.*

<sup>7</sup> *Schmidt's Jahrbuch*, 1880.

<sup>8</sup> *Zeitschrift für klinische Medicin*, 1882.

seven cases of left-sided against two right-sided affection. It also does not seem to attack the different portions of the pleura equally, for in six cases it is distinctly stated to have been more advanced on the costal pleura than on the rest of the membrane. As regards the ages of the patients all but one were middle aged or over, viz., from 35 to 62; in one only, that of Lepine, was the patient young, viz., 10 years. The symptoms during life were in most cases those of simple chronic pleuritic effusion. The microscopical appearances were in all those of alveolar, or, as it is often called, endothelial, sarcoma; and its malignant nature is shown in four cases, in which secondary deposits in the internal organs occurred; in fact, it seems to possess the same relation to the ordinary forms of sarcoma, that the infiltrating form of cancer, the *squirrhe en cuirasse* of Velpeau, does to the medullary cancer. Birch-Hirschfield, Böhme, Eppinger, and Schulz, in the description of their cases, state that the growth could be plainly seen to commence in the endothelium of the lymphatic vessels; the last-named author gives an especially minute account of his case, in which the endothelium could be seen to enlarge, divide, and finally fill the vessel with large epithelial-like cells. Böhme's description is very much similar, excepting that he gives two alterations as occurring in the endothelium, viz., an hypertrophy, in which the cells were simply larger, and an hyperplasia, in which they were increased in number. In my case I was unable to trace their origin from the lymphatic vessels; only here and there, and then mostly in the diaphragmatic pleura, could a vessel be seen filled with cells; and in none of the sections could I see the branching structures described by these authors, excepting perhaps in those taken from the lungs, and these nodules were evidently secondary to those on the costal pleura. The appearance of the cells, either isolated or in short rows, as described, seems to point most distinctly to their origin from the connective tissue cells, and this is further borne out by the structure of the larger nodules, for it is difficult to see how a so fine meshed alveolar structure could be produced had the disease begun in the lymphatic vessels. That the cells thus formed may rapidly find their way into these vessels, and there multiply or even infect the endothelial cells, is extremely probable, and is supported by their

occasional appearance in the vessels of the diaphragmatic nodules. The reason of this difference between my results and those of the other observers may, I think, be very well accounted for by the difference in the stages of the respective cases—their's being advanced, mine being comparatively recent.

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#### EXPLANATION OF PLATE XV.

Fig. 1. Section from a nodule on parietal pleura,  $\times 80$ ; *a*, fissure like alveolus; *b*, fine fibrous band in alveolus; *c*, capsule.

Fig. 2. Section from same nodule pencilled,  $\times$  about 250.

Fig. 3. Section from a nodule on parietal pleura, taken from the edge of the growth,  $\times 400$ ; *a*, rows of small round cells; *b*, the same cells enlarging; *c*, epithelioid cells forming a group; *d*, epithelioid cells in rows.

INFILTRATING CARCINOMA OF BREAST. By GILBERT  
BARLING, M.B., F.R.C.S. (PLATE XV.)

E. T., a female, aged 46 years, was admitted to the General Hospital, Birmingham, on January 17, 1882, under the care of Mr. T. H. Bartleet, by whose kindness I am enabled to report the case. The patient was an anæmic, worn-looking woman, coming to the hospital for relief from a painful enlargement of the left breast. On examining this part it was found to be considerably larger than the right; it was smooth and elastic, no tumour could be defined in it, and manipulation conveyed the impression of a generally inflamed breast; the skin was red and cedematous, adherent over a small patch towards the outer part; the nipple was retracted, and the whole breast moved freely on the deep tissues. Examination of the axilla revealed some glands, slightly enlarged, extending high up towards the root of the neck.

The patient's previous history was that of fair health until the commencement of her present trouble, fifteen weeks before admission, when she sprained her left pectoral muscles, and suffered pain in the breast as a consequence. Five weeks later she noticed a hardness round the nipple, and a curdy white discharge from the latter, the hardness extended and the pain increased, being felt soon in the axilla. All these conditions became aggravated until the time of admission. Patient had been married four months, was still menstruating, and knew of no hereditary taint of malignant disease. After consultation Mr. Bartleet determined to explore the breast, and, in the absence of inflammatory products, to remove the tumour and the glands. This was done on January 28th; the whole of the breast, the pectoral fascia and the muscles, and a wide strip of skin, including the nipple, were removed, also a handful of glands, some of which extended beneath the clavicle. The patient did very well at first, but healing was later very protracted, owing to the free removal of skin, and before it was completed there was most extensive recurrence in a complete circle around the edge of the incision, the recurrence extending rapidly over the chest wall, the whole presenting a brawny, cedematous condition, with dusky

redness of the overlying skin. Death took place in June, outside the hospital, but no *post-mortem* could be obtained. On examining the removed organ it was not possible to recognise any defined growth; there was simply seen a general enlargement of the breast, of moderately firm consistence, without cysts or degeneration, and not resembling either the hard or soft carcinomata generally met with in that gland. The skin was affected only where it was adherent, and examination of the circumference of the growth showed that the disease had been completely removed.

The accompanying drawing, kindly made for me by Dr. Saundby, shows that the growth consists principally of alveoli, packed with very large epithelial cells (A in the drawing), many of them multi-nucleated, the spaces being generally close together; in addition there were some acini, remnants of the gland tissue probably (B in the drawing), surrounded by small celled infiltrations, and containing epithelium, which contrasts remarkably in size with that above referred to (A) (Plate XV. fig. 4).

There are several points of interest about this case; its clinical aspect suggested that it might be an inflammatory condition; on microscopical examination, after removal, one still doubted whether it might not really be so; the rapid occurrence of the growth after the supposed injury is notable, as is the excitement the breast tissue would be subjected to by her recent marriage, at an age when carcinoma so commonly originates. The recurrence was what one might call terrific, both in its rapidity and its extent. I have used the term "infiltrating carcinoma" because it expresses better than any other the general and microscopical characters of the growth, although I may lay myself open to the charge of using unnecessary definitions when speaking of carcinoma as "infiltrating."

OBSERVATIONS ON THE DIAMETERS OF HUMAN  
VERTEBRÆ IN DIFFERENT REGIONS. By R. J.  
ANDERSON, M.A., M.D., *Demonstrator of Anatomy, Queen's  
College, Belfast.*

THE measurements of the bodies of vertebræ were made with a view to determine their relations in various regions, and to the diameters in other regions.

The vertical diameters of the cervical region in front and behind were generally the same—a fact generally recognised. In the dorsal region the anterior vertical diameter is generally less than the posterior. The difference is less above. In the lumbar the anterior diameter may be equal to, greater, or less, than the posterior, always excepting the last. The diameters were measured with a millimetre rule. Most anatomists recognise the fact that the lumbar curve is mainly due to the intervertebral cartilages, but the actual in the anterior and posterior diameters are given differently by different authors. The vertical diameter (anterior) increases from the 3rd cervical to the last lumbar 14 m. to 29 mm. (Henle).<sup>1</sup> The posterior vertical does not maintain the same steady increase in the dorsal and lumbar regions as the anterior. In the lumbar region a diminution is observable. Thus, in the mean of thirty measurements, the posterior vertical diameter of the second gives 27·4 millimetres and 22·2 in the 5th lumbar. And in the mean of fifty measurements the sum of all the anterior diameters in the lumbar region does not exceed greatly the posterior sum, and this although the anterior diameter of the 5th lumbar exceeds greatly the posterior.

The antero-posterior diameters increase from the 3rd cervical to the 3rd lumbar. But in the cervical region this increase is much slower than in the dorsal region, whilst in the lumbar region the antero-posterior diameter of the fifth and sometimes of the fourth are less than the third. The means of two series of measurements are given. In the first series the antero-posterior was taken from the anterior part of the body to the

<sup>1</sup> Knochenlehre, p. 37.

anterior part of the vertebral foramen. In the second series the measurement was made from the anterior part of the body to the line drawn across the points where the arch rises from the body. The latter measurements differ but little from the former in the cervical and lumbar regions but much in the dorsal.

The maximum transverse diameters vary in position in different regions. They are placed farther back in the upper dorsal region, and approach the middle of the antero-posterior diameter as they are followed upwards or downwards.

The antero-posterior and transverse diameters are most nearly equal from the 5th to the 8th dorsal vertebræ; above and below the antero-posterior are much less.

In the mean of the transverse the 5th dorsal has the smallest. A gradual increase takes place from the 2nd cervical to the 2nd dorsal vertebra, then a diminution to the 5th dorsal, and this is succeeded by an increase which goes on to the 5th lumbar.

In certain parts of the dorsal region in most of the vertebral columns examined, the bodies were found not to be equilateral. As a rule, the right sides of the bodies are greater than the left, from the 7th to the 10th dorsal vertebræ. This may hold for all the vertebræ lying between the 3rd and 11th. Sometimes, with a large right side from the 7th to the 10th, a large left side is present in the upper dorsal region.

This unilateral enlargement is probably due to the lateral curvature. The greater weight of the viscera on the right side is, without doubt, an element in these cases. In the former case the bodies of the vertebræ are at the right sides less curved, and the antero-posterior diameter of the bodies are placed a little to the right of the median line.

The transverse diameters of the bodies of the dorsal vertebræ of most animals that I have examined exceed the antero-posterior. The exact relations I hope soon to publish.

In the following table will be found:—

- A. The vertebral column of an adult where the largest measurements were found.
- B. The vertebral column of an adult where the least measurements were found.
- C. The means of the transverse diameters of the bodies of fifty-three vertebral columns.

D. The means of the antero-posterior diameter of the bodies of twenty-eight vertebral columns.

E. The mean of antero-posterior diameters of twenty-five vertebral columns.

F. Anterior, and

G. Posterior vertical diameters in different regions.

A table is added with the mean of collective heights.

The measurements given above point to the following conclusions :—

- (1.) The anterior vertical diameters of the vertebræ, from the 1st dorsal to the 3rd lumbar inclusive, are less than the posterior.
- (2.) The anterior vertical diameter of the 4th lumbar is equal to its posterior, and in the 5th it is greater.
- (3.) The transverse diameters of the bodies increase from 2nd cervical to 2nd dorsal, then diminish to 5th dorsal, then increase to last lumbar.
- (4.) The antero-posterior diameters increase gradually from 3rd cervical to 3rd lumbar, and then diminish.
- (5.) The non-equivalence of the lateral parts of the bodies is generally observable from the 7th to the 10th dorsal, but may extend to the 3rd and 11th vertebræ.

[TABLE



## 344 DIAMETERS OF HUMAN VERTEBRÆ IN DIFFERENT REGIONS.

TABLE.

	A.						B.			C.	D.	E. Antero- Posterior Diameter, XXVIII.- LII.	F.	G.
	T.	A.	H.		T.	A.	H.		Vertical Diameter of I.-XXXI., omitting XXX.					
			Anterior.	Posterior.										
Cervical 2.	21	17	a. 22 b. ..	14	14	a. 18 b. ..	19.0	15.6	15.4	19.5	..			
" 3.	23	16	13 ..	18	14	13 ..	19.9	15.2	14.9	12.4	..			
" 4.	23	15	13 ..	19	13	12 ..	21.7	15.7	15.4	12.2	..			
" 5.	25	16	15 ..	19	14	12 ..	23.0	16.2	15.0	12.1	..			
" 6.	30	18	12 ..	21	15	11 ...	25.2	17.8	16.4	11.5	..			
" 7.	30	19	14 ..	23	14	12 ..	27.5	18.3	16.9	13.0	..			
Dorsal 1.	31	17	16 17	25	14	14 16	27.9	17.3	17.4	14.8	15.9			
" 2.	30	19	18 19	23	17	17 18	28.2	17.6	18.9	16.8	17.4			
" 3.	31	22	17 19	24	15	15 18	26.3	19.6	20.5	17.6	18.2			
" 4.	29	27	18 20	22	19	18 20	26.0	22.0	23.2	17.8	19.2			
" 5.	30	28	19 22	23	22	16 20	25.9	24.2	23.9	17.8	20.2			
" 6.	30	30	20 23	25	23	18 20	27.4	25.6	25.3	18.1	19.9			
" 7.	32	31	20 25	26	25	18 21	28.8	26.8	26.5	18.5	20.6			
" 8.	33	33	22 25	27	25	18 22	30.1	28.2	28.3	18.9	21.6			
" 9.	34	35	22 25	28	26	19 22	31.9	29.4	28.2	19.7	21.9			
" 10.	40	37	23 25	30	27	22 23	34.1	29.4	29.4	21.0	22.9			
" 11.	45	37	23 28	32	27	22 24	36.5	29.8	29.8	21.9	24.9			
" 12.	45	35	23 29	33	25	25 26	40.5	30.0	30.8	23.6	25.4			
Lumbar 1.	51	37	25 29	37	25	25 27	42.2	29.9	31.1	24.6	26.5			
" 2.	53	43	26 28	40	26	25 25	44.0	31.9	31.2	25.9	27.4			
" 3.	57	44	28 28	43	29	25 25	47.7	36.8	32.2	26.7	27			
" 4.	57	43	26 26	43	27	26 22	48.3	33.8	31.4	26.1	26.0			
" 5.	63	44	30 24	43	29	27 20	52.7	36.5	31.3	27.2	22.2			
T.—Transverse diameter. A.—Antero-posterior. H.—Vertical diameter. { a. antero- b. posterior.								MEAN OF HEIGHTS.						
								Anterior.			Posterior.			
								80.0			..			
								222.3			245.0			
								132.9			123.3			

NOTE ON A SIMPLE FORM OF LIPPMANN'S CAPILLARY ELECTROMETER USEFUL TO PHYSIOLOGISTS.<sup>1</sup> By Professor JOHN G. M'KENDRICK, M.D.

THE action of the substances set free on the electrodes in electrolytic decomposition, and the energy shown as motion in these circumstances, is strikingly manifested by the behaviour of a drop of mercury in dilute sulphuric acid, when the positive pole of a battery is put in connection with the mercury, and the negative pole dips into the acid. The mercury extends towards the negative electrode during the passage of the current, becoming covered with a film of sub-oxide, which dissolves in the acid, leaving a bright surface. By making and breaking the current a series of oscillations is set up. Movements in the mercurial electrode and adjacent acid have been observed by Henry, Gerboin, Erman, J. F. W. Herschel, Draper, Paalzow, Faraday, and Quincke,<sup>2</sup> and they received various explanations. Erman,<sup>3</sup> in 1809, was the first to observe that when a drop of mercury was placed on a grooved surface between the electrodes it moved towards the negative pole; and he also observed that "a drop of mercury in a horizontal tube, with dilute acid on both sides, moved at the passage of the electric current through the tube towards the negative electrode."<sup>4</sup>

This latter phenomenon was fully investigated by Lippmann,<sup>5</sup> and led, along with researches by Quincke, not only to a theoretical explanation of electro-capillary action, but also to the construction of the capillary electrometer. It is now known that these phenomena, both as seen in the experiment with the globule of

<sup>1</sup> Read before the Philosophical Society of Glasgow, January 17, 1883.

<sup>2</sup> For an historical account of this subject see Lippmann, *Annales de Chimie et de Physique*, 1875, p. 540. Also references in arts. "Capillary Action, by Professor Clerk Maxwell, *Encyclop. Britann.*, vol. v. p. 65; and "Electrolysis," by Mr. W. N. Shaw, vol. viii. p. 108. See also art. "Electricity," Watt's *Dict. of Chem.*, third suppl., vol. viii. part i. p. 714.

<sup>3</sup> Gilbert's *Annalen*, t. xxxii. p. 261, 1809.

<sup>4</sup> Art. "Electrolysis," *op. cit.*

<sup>5</sup> Lippmann, *Comptes Rendus*, 1873, p. 1407; *Annales de Chimie et de Physique*, 1875, p. 494; Pogendorff's *Annalen*, cxlix. p. 547; also translated in *Phil. Mag.* [4], xlvii. p. 281.

mercury and in the capillary tube, are due to a change in the surface tension produced by the electrical polarisation of the surface of the mercury.

Lippmann's form of the electrometer consists of a tube of ordinary glass, 1 metre long and 7 millimetres in diameter, open at both ends, and kept in the vertical position by a stout support. The lower end is drawn into a capillary point, until the diameter of the capillary is  $\cdot 005$  of a millimetre. The tube is filled with mercury, and the capillary point is immersed in dilute sulphuric acid (1 to 6 of water in volume), and in the bottom of the vessel containing the acid there is a little more mercury. A platinum wire is put into connection with the mercury in each tube, and, finally, arrangements are made by which the capillary point can be seen with a microscope magnifying 250 diameters. Such an instrument is very sensitive; and Lippmann states that it is possible to determine a difference of potential so small as that of the  $\frac{1}{10000}$  of a Daniell. It is thus a very delicate means of observing and (as it can be graduated by a compensation-method) of measuring minute electro-motive forces.

As the sensitiveness of the instrument depends essentially on the smallness of the bore of the capillary, and on the constant electro-motive force between the mercury and the sulphuric acid (termed by Lippmann the *différence électrique*), it is important to have the tube as fine as possible, and also to prevent variations in the strength of the sulphuric acid by evaporation. These considerations led to improvements in the construction of the instrument, thus described by Burdon Sanderson: <sup>1</sup>—"Two important improvements have been recently made in the construction of this instrument by Professor Lovén,<sup>2</sup> of Stockholm. The first of these consists in constructing the tube which contains the dilute sulphuric acid of extremely thin glass, so that the capillary tube which is in contact with its internal surface can be observed with

<sup>1</sup> Burdon Sanderson "On the Electro-motive Properties of the Leaf of *Dionæa* in the Excited and Unexcited States," *Phil. Trans.*, part I., 1882. See also his lecture "On the Excitability of Plants," at the Royal Institution of Great Britain, 9th June 1882. In stating his opinion that "it is much better to measure the electro-motive value of each excursion with the aid of the compensator, than to deduce it, as has been done by Fleischl, from the 'compensation pressure,'" Dr. Burdon Sanderson refers to Fleischl's paper, *Archiv f. Anat. u. Physiol.*, 1879, p. 269.

<sup>2</sup> Lovén, *Nordiskt Medic. Arkiv.*, vol. xi. No. 14.

a magnifying power of at least 300 to 400 diameters (No. 10 Hartnack). The other consists in cementing the sulphuric acid tube to the mercury tube, so that no evaporation takes place. By the first the instrument is rendered much more sensitive; by the second, variations in the strength of the sulphuric acid which arise from evaporation are avoided."

The instrument thus described is difficult to make, and requires delicate handling. Whilst studying the subject, and endeavouring to see the phenomena for myself, I resorted to the simple expedient of taking a bit of glass tubing, 60 millimetres in length, and 2.5 millimetres in internal diameter, bending one end at a right angle, 20 millimetres from the end, and the other end at a right angle, 6 millimetres from the end, and then drawing out the tube to a capillary in the blow-pipe flame. The diameter of the capillary tube varies from .02 mm. to .001 mm. I then slip a bit of thick-walled india-rubber tube on one end of the glass tube, and exhaust the other end of the india-rubber tube with a powerful syringe. Having some clean mercury in a beaker covered with a deep layer of dilute sulphuric acid (1 to 20 of water by volume), I first immerse the end of the glass tube into the mercury, and then exhaust by the syringe. When mercury appears in the upper portion of the tube, I raise it a little so as to cause it to dip for an instant into the dilute acid, and then I dip into the mercury a second time and draw a little more of it into the tube. Thus, with a little practice, I succeeded in filling tubes, so that there is a reservoir of mercury at each end, with a little sulphuric acid in the centre of the capillary. This is placed on a suitable platform on the stage of the microscope, and a thin platinum wire is dipped into the mercury at each end.

Such an instrument is very sensitive, showing the muscle-current, the negative variation, and the changes in potential in the injured heart of the frog whilst beating. As observed by Burdon Sanderson, it is striking to notice "that the movements of the mercurial column not only correspond closely in time to the actual changes which they represent, but express with very great accuracy the differences of a potential which actually exist in each successive phase of a variation."

It will be found that, in the instrument I have described, the

column of mercury will not always return to zero when the electro-motive force pushing it in one direction has ceased to act. When it does return, the return is due to the pressure of the column of mercury against which the force acted. As a matter of fact, the most sensitive instruments are those which do not return to zero, but in which the movement of the mercurial column corresponds to each change in the direction of the electro-motive force, or to any increase or diminution of the electro-motive force even in one direction. Thus, if there be a negative oscillation, it will be at once seen, although it last for a fraction of a second. Sometimes the instrument is "sluggish" in its movements. This is caused by the mercury, or the acid, or the tube not being perfectly clean.

An electrometer such as this can have its deflections photographed<sup>1</sup> or exhibited on a screen, whilst its simplicity and cheapness makes it possible to place a sensitive instrument in the hands of every student—or, better still, a student may make it for himself.<sup>2</sup> For convenience in working, when it may be necessary to make observations with a galvanometer at the same time, the image of the capillary may be thrown on the plate of a camera, adjusted to a microscope, and thus the trouble of looking through the latter is got rid of.

<sup>1</sup> Marey obtained such a photograph in his research on the Electrical Discharge of the Torpedo (*Physiologie Experimentale*, 1877, p. 33, fig. 19), and Burdon Sanderson has photographed the oscillations caused by the changes of potential in *Dionæa*, and in the frog's heart.—Royal Institution Lecture, fig. 10.

<sup>2</sup> Various forms of the instrument were shown, and the oscillations of the mercury were projected on a screen, the oscillations being magnified 300 diameters. There is nothing novel either in the theory or in the mechanism of this instrument, as it in no way differs in principle from one shown at the meeting of the British Association in Glasgow in 1876, by Professor Dewar of Cambridge, but I have shown an easy and convenient mode of making such an instrument of exquisite delicacy for physiological purposes. A figure of Professor Dewar's instrument is shown in Dr. S. P. Thompson's recent Manual on Electricity.

ON SO-CALLED SPONGE-GRAFTING. By KENDAL FRANKS,  
M.D., F.R.C.S.I., *Surgeon to the Adelaide Hospital, Dublin,*  
&c., and P. S. ABRAHAM, M.A., B.Sc., F.R.C.S.I., *Curator to*  
*the Museum, and Member of the Court of Examiners, Royal*  
*College of Surgeons in Ireland.*<sup>1</sup> (PLATE XVI.)

THE idea of treating certain kinds of wounds accompanied by loss of substance, as well as ulcers, by means of a method called sponge-grafting, was first brought before the profession by Professor D. J. Hamilton. In an interesting and valuable paper, published in the *Edinburgh Medical Journal*, November 1881, he gives an account of his method of proceeding, and details the results he obtained in five cases in which he applied it to the human subject, as well as in certain cases in which he observed the effects of inserting pieces of sponge into the living tissues of rabbits.

The able manner in which Dr. Hamilton has introduced this subject, and the clinical success which has followed his method, solicit a further trial at the hands of surgeons, the more so that this method opens up new fields of investigation in the domains of surgery as well as in those of pathology and histology. It is not suggested as a new-fangled and empirical method of treatment to oust all former therapeutic means; it is the result of careful thought, and it fulfils an object which certain conditions require. When a wound is made in any part of the body, if unaccompanied by loss of substance, our object is generally to procure a healing of the wound as rapidly as possible. But if we find at the same time that a loss of substance exists, cicatrization is not enough; it would in general be far better if we could ensure the restoration of the part which has been destroyed, a reformation of the tissue which has been lost. Sometimes, no doubt, an ulcer may heal level with the skin, but oftener it cicatrises, leaving a distinct and unsightly depression. What this new method proposes to do, in cases where there

<sup>1</sup> Most of this paper has been in MS. since July 1882. Dr. Franks is mainly responsible for the clinical part, and Mr. Abraham for the histological details.

exists a loss of substance, is to afford a scaffolding in which the new tissue can be built up, and this scaffolding is supplied by a fine porous substance like sponge. There are no special inherent properties in the sponge itself which render it essential to the process ; it takes no active part in the growth of tissue ; its rôle is purely a passive one. Dr. Hamilton proposes it as the material which hitherto proves itself the most suitable for the purpose, and up to the present we have no better substitute to offer.

When a sponge is placed on a granulating surface, it soon becomes fixed there, and it is now beyond doubt that new tissue forms in the interstices of the sponge. The manner in which this new tissue forms has been studied by Professor Hamilton, and the startling histological conclusions at which he has arrived by microscopical examination of the pieces of the excised grafts, seem to court further investigation.

It should here be stated that it has not yet been demonstrated whether different kinds of tissue can be reproduced in this way. Up to the present the cases in which it has been tried have shown the formation of granulation tissue, but whether under certain conditions this granulation tissue may not subsequently be transformed into the same material as that which surrounds the sponge, is a question which demands further inquiry. We have had a case under observation of necrosis of the femur, where, as soon as the sequestrum was removed, a piece of sponge, properly prepared and rendered aseptic, was placed into the space left by the sequestrum. We have also had a case where a sponge had been for some weeks imbedded in the muscles of the thigh, and which to all appearance became incorporated. It will be interesting to observe whether in such cases the material which has formed in the sponge meshes will eventually develop into bone and muscle respectively.

Since the appearance of Dr. Hamilton's paper very little has been published on this subject.

In the *British Medical Journal* for May 13th last, Dr. M'Ewen is reported to have shown some microscopical specimens to illustrate the process of "sponge-grafting" before the Glasgow Pathological and Clinical Society. He had grafted one-half of an ulcer on the outer side of the leg, but the result

did not come up to his expectations, and he did not therefore "find the process of any value." "The sections shown demonstrated the fact," says the report, "that absolutely no change had taken place in the sponge, and Dr. M'Ewen therefore objected to the term 'sponge grafting,' because the sponge tissue never became alive."

We hope to show presently that very important changes do occur in the sponge, and that as our own cases, as well as several others, prove, it is ultimately completely absorbed. Dr. Hamilton, moreover, never claimed that the sponge became alive, or that it acted otherwise in the body than would any other dead animal tissue. There is indeed no growth of the sponge itself, and on this account the term "sponge-grafting" is no doubt defective.

*Case I.*—The first case in which we tried the process was in that of an old woman, aged 70, who was admitted to the Adelaide Hospital, and came under surgical treatment on the 10th February 1882, for a large ulcer occupying the lower middle fourth of the right leg. The ulcer had inflamed elevated edges. The surface of the ulcer was occupied by a large slough which, when it separated on the 27th of the same month, exposed the muscles of the calf. The leg all round the ulcer had an inflamed erysipelatous-looking blush. Ordinary methods of treatment were employed until the 17th of March, when the first piece of sponge was grafted. It was not cut so as to cover the whole ulcer, but a circular piece, one inch in diameter and about half an inch in thickness, was used. On the third day it felt firmly adherent. A few days later five smaller pieces were grafted in different parts of the ulcer. They all became firmly adherent in a few days.

These sponges were all removed at different periods; one on the eighth day, one on the fourteenth, one on the thirtieth, and the remainder on the forty-eighth day. Sections of these sponge-grafts, some of which have been photographed by the kind assistance of Dr. R. Hayes, will be referred to presently.

These experiments have served to supply us with material for histological and pathological study; we shall presently see that the results observed during the period the sponges were allowed to remain in the tissues are of much surgical importance.

*Case II.*—The next case in which this method of treatment was adopted was in a boy aged 9, who suffered from necrosis of the lower portion of the right femur. As part of the sequestrum had separated, an incision was made about five inches long on the outside of the thigh in its lower half, and parallel to the femur. This incision, of course, reached down to the bone in the greater part of its extent. The sequestrum was removed. Into the lower angle of the wound a



sponge was placed on June 15, 1882; it was made wedge-shape, so as to reach down from the surface of the skin into the cavity in the bone. It measured one inch and a half in length, three-quarters of an inch in depth, and three-eighths of an inch in width. The next day it was adherent, and granulations could be seen making their way into it from each side. It rapidly filled with new tissue, and only a small central oval piece on the surface could be recognised after a month. The margins had become covered with new skin. Later on it completely disappeared. A piece of sponge placed in the upper angle of the wound on June 24th was considerably larger, and measured two and a half inches in length, one inch in depth, and half an inch in width. It was also made wedge-shaped, and reached from the bone to the level of the skin. It became adherent the next day, and seemed to fill rapidly with granulations. In a short time, however, it began to rise above the skin level, and in consequence it was trimmed down. In another week it had again transgressed the surface, and the trimming process was repeated, the operation being accompanied by bleeding. On July the 18th the piece seemed much loosened in the wound, and long bridges of granulations could be seen running from the sides into the sponge. It was then removed, the boy being placed under the influence of chloroform for a few minutes, whilst a sharp pair of scissors divided the granulations at their base. This was done, (1) because the graft did not promise to be clinically successful, and (2), because it promised to afford a good opportunity of investigating the process taking place on both sides of the sponge simultaneously. This excised piece of sponge was prepared for microscopical examination in the same way as the former ones.

*Case III.*—A cook, aged 22, was admitted to the Adelaide Hospital on December 28, 1882, suffering from an ulcer about the size of a halfpenny on the inner side of the left leg, and a smaller one higher up on the calf about the size of a threepenny bit. Both were circular and punched out, with unhealthy sloughy bases. She attributed them to constant standing in the kitchen, and had suffered from them for over three months. Astringent lotions had been tried, and constant bandaging, but without effect. The discharge was scanty and of a reddish-brown colour. There was no history or evidence of syphilis. The larger ulcer was grafted on January 3rd last. The sponge, which was prepared in the usual manner, was an inch in diameter, and about one-eighth of an inch in thickness. In two days it was firmly adherent, and in a few days bled when pricked. On the ninth day the ulcer had contracted to the size of the sponge. On the twenty-fourth day the centre of the sponge was invisible, being hidden by the granulations which had made their way through the sponge and appeared on the surface. Four days later this island of granulations was the size of a fourpenny piece, and was surrounded by a circle of sponge. Eight days later, only a small ring of sponge remained; three skin-grafts were then placed on the surface, within the ring, and when examined three days afterwards, one was found to have acted in

a manner similar to that observed in ordinary cases of ulcers when skin-grafts are applied. A few days later the graft measured two lines in diameter.

*Case IV.*—A woman, aged 60, was admitted to the Adelaide Hospital on January 12, 1883, suffering from an irregular-shaped ulcer on the inside of the right leg, measuring upwards of three inches in diameter. The edges were elevated and indurated. The base of the ulcer was very unhealthy and callous. The skin all round the ulcer was brawny and livid. She had suffered on and off for nearly thirty years from this condition. The exciting cause was evident, as she had several large varicose veins, tortuous and prominent, a little below the knee. The day after admission three pieces of sponge were placed on the surface of the ulcer. The largest piece, circular, about one inch in diameter and a quarter of an inch thick, was placed near the centre. Two smaller pieces, about three quarters of an inch long, a quarter of an inch wide, and one-eighth of an inch thick, were placed, one above and one below the central piece. They were all adherent the next day. The immediate effect on the ulcer was very remarkable. It assumed in a few days a much healthier appearance. The edges gradually lost their hard elevated character, and cicatrisation proceeded rapidly round the circumference. On the 25th the ulcer was not more than half its original size, and the smaller sponges were becoming rapidly incorporated. One of them became detached to a very slight extent, being partially undermined by the new epidermis encroaching upon it from the margin of the ulcer, but the rest of this piece became almost unrecognisable from the surrounding granulations. The larger piece of sponge required to be cleaned every day, as pus collected in its upper portion. It has not yet revealed granulations on its surface. The veins were now treated radically, by passing acupuncture needles beneath them, and dividing the veins between two of these. This has so far not materially affected the process going on in the ulcer or sponges.

We have employed "sponge-grafting" in several other cases besides those detailed, but as they revealed no points of especial interest which have not been illustrated by the cases recorded, we have not reproduced them here.

A patient of Dr. Kelly, of Jervis Street Hospital, Dublin, had been operated upon for hare-lip. All the tissues included between three needles and the "figure of eight" sutures had sloughed, leaving a most formidable chasm in the lip. A piece of sponge was fitted into the cavity and maintained in position by flexible collodion. Dr. Kelly thus writes, under date August 10 :—"My sponge only declared off yesterday, and I write to tell you the result. As you are aware, cicatrisation extended under it rapidly, but did not displace the graft completely until a very remarkable result was produced. When the sponge finally separated I could perceive hardly a trace of a depression, and better still, the patient and her friends are charmed by a result which I awaited in fear and trembling."

Dr. Kelly informs us that he has since employed the method in two cases of loss of tissue of the fingers, with eminently satisfactory results.

Dr. Robert M'Donnell has kindly given us the particulars of a case which occurred many years ago in Steevens' Hospital in Dublin under the care of the late Dr. Hutton, and which is of great interest in its bearing upon the subject under discussion. Dr. Hutton had excised a large hydrocele of the neck. Shortly afterwards a sudden and profuse, probably venous, hæmorrhage occurred. In the absence of Dr. Hutton the surgical resident plugged the wound with a sponge and thus controlled the bleeding.

The sponge was not removed owing to a fear of the hæmorrhage recurring. Gradually the wound closed up over the sponge, and completely healed. The sponge was never seen again. There can be scarcely any doubt now, in the light of recent observations, that the sponge was absorbed.

It was formerly a not uncommon practice in the treatment of epistaxis to plug the nares with pieces of sponge. Experience then proved the importance of not allowing the sponge to remain too long in its new position without changing it. If permitted to remain *in situ* longer than three days, great difficulty was often experienced in removing it, and even before this period its withdrawal was nearly always accompanied by fresh bleeding. The same observations apply to the use of uterine sponge tents.

In all these cases the same results ensue as we observe when pieces of sponge are applied to a raw or granulating surface. When applied for a short time, forcible evulsion will tear the tissue which has permeated the meshes of the sponges; and if a longer period have elapsed considerable force may have to be used before the adhesions are torn through.

In preparing the sponges for the so-called "grafting," we have uniformly followed the method recommended by Dr. Hamilton. The sponge is first allowed to lie in dilute nitro-muriatic acid; we have generally allowed forty-eight hours for this stage. Then the sponge is washed, first in water, afterwards in liquor potassæ, and finally placed in a solution of carbolic acid (1 in 20), till required for use. Thus all silicious and cretaceous materials are dissolved out, and nothing remains but the horny framework of the sponge, which, being an animal tissue, does not act as a foreign body, but is absorbed after a longer or shorter interval, as our experience proves.

The process by which this absorption takes place, is shown in the microscopical preparations which we have been able to make

of the sponges excised at varying periods of the growth of tissue within them.

When a sponge so prepared is placed on an ulcerated surface, or into deeper tissues where a loss of substance has taken place, it probably first gets filled with lymph. The lymph then undergoes organisation, and according to Dr. Hamilton, in a manner similar to that which obtains in blood-clot.

When the granulations approach the surface in the case of an ulcer, skin begins to form, generally growing from the margins of the ulcer over the tissue in the interstices of the sponge.

One of our sections shows that the formation of epidermis may take place within the sponge itself over the new tissue which has formed within its interstices, and before the upper stratum of the sponge has become filled with granulation tissue. In case III., one of the three skin-grafts placed over the new tissue which had formed in the sponge became adherent, and materially assisted in the process of cicatrisation. The formation of skin, however, seems to take place more slowly over the new tissue so formed than over an ordinary healthy ulcer.

We have observed one effect which a sponge has when it becomes fixed on the surface of an ulcer, and which is sufficiently characteristic to call for some comment, namely, the general stimulating effect it produces on the process of repair. We have observed this in cases of unhealthy ulceration, where ordinary stimulating applications seemed to have very little, if any, effect, and where the whole character of the ulceration was indolent. As a general rule, within a few days after the application of the sponge the whole type of the ulcerative process seemed to change, the edges began to assume a healthy appearance, and the ulcer began to cicatrise. We are all familiar with the appearances which follow on skin-grafting; the delicate promontories of new cuticle which are thrown out from the sound skin towards the skin-graft. The same appearances are nearly always seen to take place in the region of the sponge which has been applied to an ulcer. This stimulating effect is so remarkable that we would recommend this process to be adopted in those cases of indolent ulceration which so often try the ingenuity and patience of the surgeon, even though the actual loss of substance be of the smallest.

The application of sponge to an ulcer sometimes fails, by reason of a sort of spontaneous amputation of the piece taking place. This we have not observed in the case of ulcers with undermined edges, when the sponge is cut level with the skin, and the edge of the sponge is slipped beneath the undermined edges of the ulcer. In these cases the skinning process seems to spread rapidly inwards from the edges over the sponge. But when the edges of the ulcer are not undermined, and the sponge stands up above the surrounding level, as cicatrization is greatly stimulated by the sponge, there is a danger that this process, gradually growing inwards from the healthy skin towards the sponge, may spread *beneath* the sponge instead of *over* it, and by its constricting power may gradually strangle the young tissue forming in the sponge, and cause finally the whole piece to slough off. This we have several times observed to take place, and this we believe to be the explanation why sometimes, in the practice of others, "sponge-grafting" has failed.

Dr. D. J. Hamilton has recently<sup>1</sup> advised the surgeon to apply successive thin layers of sponge over a wound, instead of the thick slices which he had formerly advocated. Our observations entirely agree with this change. We have frequently found it necessary to pare down a thick piece of sponge, so as to leave nothing but the thin layer which has become infiltrated with organising tissue. Our object has been twofold; first, to prevent the sponge transgressing the level of the skin around, which it has a great tendency to do; and, secondly, to remove a framework which gets filled with pus, and which may become a fertile soil for putrefactive changes.

We have never found in any of our cases any evidence of the entry of nerve fibres into the sponge. Of course it is possible that such may occur, as is illustrated in the case recently reported by Mr. Ferguson.<sup>2</sup>

The portions of sponge excised were in all cases hardened for about a fortnight in Müller's fluid, and subsequently in alcohol. The sections were cut by means of Williams' freezing microtome, and stained with picro-carmin and hæmatoxylin. It was found that the thinnest sections were difficult to manage,

<sup>1</sup> *Brit. Med. Jour.*, Jan. 6, 1883, p. 7.

<sup>2</sup> *Brit. Med. Jour.*, Dec. 16, 1882, p. 1202.

portions of the sponge not infiltrated with growing tissue being brittle and crumbling away; some of the slices were therefore stained, dehydrated, and cleared *on* the slide. In other cases staining was effected in the mass. Most of the sections were mounted in Canada-balsam, a few in glycerine. Those which were most successful were taken from the sponges of case I., which had been in the ulcer for eight, fourteen, and thirty days respectively. Others were taken from the sponge of case II., which was in the process of being pushed out after lying in the wound for twenty-four days. They all present certain characters in common, the base of the new growth being composed of an embryonic, more or less developed, connective tissue, which in the deeper parts has quite replaced the horny sponge skeleton. Above this deep part the new tissue occupies merely the interstices of the sponge, while more distally still there is to be seen the unaltered sponge containing only blood, or sometimes fibrinous deposit, with scattered blood corpuscles. In the act of cutting out the pieces of sponge for examination hæmorrhage always took place, the porous sponge and new growth becoming more or less filled with blood. Thus we may account for many of the apparent extravasations seen in the sections, and for much of the clot in the unchanged sponge.

Wherever the tissue has infiltrated, the horny fibres of the sponge are evidently fewer in number, and, as Dr. Hamilton has described, the presence of numerous giant-cells in their immediate neighbourhood is a striking feature. Broadly speaking, these sections bear out Dr. Hamilton's descriptions in many respects; but, as will soon be seen, their close examination reveals several material differences. Under a low power the most noticeable elements are the blood-vessels, which pass up from the base, sometimes with little sinuosity, and giving off but few branches; at other times with tortuous course, and inosculating freely. Their course is often at once indicated by a closely surrounding area of small cells, with darkly stained nuclei resembling leucocytes. When they approach the periphery, the branching and anastomosing is very marked (fig. 1), and the curves, whenever they appear, seem to be as often synclinal as anti-clinal. The distal parts of the new tissue are thus the most vascular; and considering, moreover, the facts

that the branches vary in diameter, and that the anastomoses take place in the meshes of a sponge, it is difficult to understand how they could have been pushed up as loops from below. Their formation *in situ*, or at any rate their growth upwards and subsequent anastomosis, appears to be more probable; and indeed there seems to be abundant evidence of the new formation of the branches at least, in most of the sections examined (fig. 2). In the older parts of the new tissue, the larger blood-vessels, when cut longitudinally or transversely, are seen to be surrounded by a finely granular, homogeneous area, sometimes almost hyaline, which passes on the one side into the cells which make up the inner wall of the vessel; and on the other, into the matrix, and into the cell substance of the cells of the tissue around—forming, in fact, an adventitia. No such differentiation has taken place in the youngest growth, where the wall appears to be formed by a condensation of the surrounding cellular tissue. The cells nearest to the lumen are more closely packed, but they seem to pass gradually, and without much change of form or character, into the larger cells of the neighbourhood. The cross section of these capillaries sometimes shows the lumen to be more or less occupied by a collection of cells other than blood-corpuscles, which are united by processes to the cells of the capillary wall, and which show signs of multiplication and division of their nuclei. The smaller of these cells appear identical with colourless blood-corpuscles, the larger with the cells of the wall and of the surrounding tissue. Do these facts not suggest another possible mode of capillary formation? The formation of capillary branches is frequently to be observed in the younger as well as in the older growths. Sometimes a heaping-up of the cells forming the wall is seen, from which the new branch is evidently sprouting (fig. 2, *a*); or a line of cells may be seen springing from the wall and uniting with cells of the general tissue, or stretching across to a neighbouring capillary.<sup>1</sup>

In the sponges which had been but a comparatively short time in the wound, the deeper portion of the new tissue consists mostly of rounded, cuboid, or irregularly-shaped cells, which

<sup>1</sup> Figures illustrating some of the above points are given in a paper "Notes on the Vessels of New Growths," in the *Transactions of the Academy of Medicine of Ireland* for this year.—P. S. A.

vary also in size and in the relative amount of nucleus and cell substance. The prolongations which the latter sends out, and which join with processes from neighbouring cells and with the tissue matrix, are usually less developed than in the case of the older growths. Some of the smaller cells have a large, darkly-staining nucleus, and are hardly distinguishable from ordinary leucocytes or pus-corpuscles; others have the nucleus more faintly coloured, and there are various gradations of division of the nucleus. With high objectives, the reticular structure of the cell substance, and particularly of the paler nuclei, is quite apparent. The cells of the older tissue also present innumerable differences in size, formation, and structure. The fibrillation of the cell substance, and its passage by processes into the substance of other cells, and into the material between these, is still better seen in the highly magnified older parts (fig. 3).

At the distal or youngest part of the new growth, the cells are so closely packed, and their nuclei for the most part stained so deeply, that there is generally formed a more or less distinct bordering area, which looks under a low power like a layer of leucocytes. There may be a few of the latter present, but with a high power and in thin sections the cells are seen to be rapidly multiplying connective tissue cells, identical with those above described.

Even with an enlargement of forty diameters, the giant-cells can be easily made out. They often occupy the angles between the branching keratode fibres, wrapping around them, or lying closely apposed along their sides. Now and then they are to be observed capping the end of a fibre, and fitting into irregular cavities, which the extremity may show (figs. 4 to 9). In one or two cases only could any indentation be found on the side of the fibre (fig. 5, c). The thinning and pointing of the fibres which Dr. Hamilton mentions is not as a rule so evident in these sections (see, however, fig. 8); nor is there any special striation of the fibre.

That these giant-cells act as "spongo-clasts" is probably as well supported by the appearances observed as in the case of the "osteo-clasts" of bone. It seems likely, however, that if they be the active eroders, the erosion is mainly effected at the ends of the fibres. Occasionally a fibre appears to be softening and



breaking down at its extremity, but even then a giant-cell is always to be found in the immediate neighbourhood.

The giant-cells vary very much in size and shape, and also in construction. The largest ones contain numerous nuclei (figs. 4 and 5) which may be irregularly disposed throughout the mass, or arranged more peripherally; while the smaller sometimes contain but a single nucleus (fig. 5, *d*). Rarely the nuclei are so numerous that but little other substance is left, and then the giant-cell appears as though it were made up of an aggregation of small cells. The texture of the cell in some of them has a more granular appearance than in others, and it is generally better seen in the doubly-stained preparations. With a one-sixth objective and No. 2 eye-piece the granules are not usually resolvable in a network, but with a one-sixteenth oil immersion (Leitz) this structure is plainly indicated. The nuclei are for the most part oval in shape, and they do not stain deeply—thus resembling the larger cells of the neighbouring tissue. Their size is tolerably uniform, but sometimes a few are seen smaller than the rest. The nuclear reticulum is generally distinct, and occasionally there is evidence of a karyokinesis.

In fig. 10 is shown a cell which at one or two points gives off processes, attaching it to neighbouring cells; it is seemingly nothing more than one of the larger connective tissue cells which has increased in size, and has become more apparently granular. Multiplication of its nucleus would give us an ordinary giant-cell; and indeed every stage is to be observed from the common connective tissue corpuscle, up to the largest giant-cell. This fact does not, however, preclude the possibility of other methods of production for giant-cells. As mentioned above, the substance of the cell sometimes passes, without line of division, into the intercellular matrix of the connective tissue around; and it is difficult to resist the conclusion that a portion of this may not assume an individuality, and acquire nuclei perhaps by migration and subsequent multiplication. The appearances, indeed, observed in these cases, as in some others, are not opposed to Stricker's new views on the relation of ground substance and corpuscular elements in tissues.<sup>1</sup>

The skinning over of the sponge has commenced in the piece

<sup>1</sup> Ashhurst's *Encycl. Surg.*, vol. i. part i.

which had been eight days planted, and it has still further proceeded in the older pieces,—generally extending along the top of the granulation tissue irrespective of the sponge fibres, which become imbedded in the new growing skin. The growth of epidermis coming in from the sides can thus be well studied in these sections. Where it is thickest, all the elementary epidermic layers are sometimes present, but generally one or more are imperfectly developed. The uppermost stratum of the youngest epidermis, instead of consisting of horny scales, is generally made of large flattened nucleated cells, the deeper ones being greatly swollen, vacuolated, and containing nuclei showing indirect division. Air spaces are also apparent between the cells. Sometimes the large dividing nucleus is in the middle of an apparently empty space, and occasionally the new nuclei seem to be invested with cell substance,—a process looking like an endogenous formation of cells. The smaller scattered cells with dark nuclei, which are sometimes seen between the larger ones, are possibly thus formed. It is by extension of this layer that the skin appears to grow into the sponge. Whether any of the branching cells of the granulation tissue are transformed into these epidermic cells, cannot be satisfactorily made out in the specimens—but the suspicion of such a possibility has arisen. As a rule, however, the advance of the epidermis appears to be by division of the cells of the layer just alluded to. In the thicker circumferential portion of the epidermis, a well-marked stratum lucidum is shown, which becomes gradually lost above the stratum just described. Just below the lowest columnar cells of the rete Malpighii, a well-defined layer of dark minute granules is to be seen in these sections. They look like finely comminuted “debris;” and in places, and with a high power, they can be made out to be in organic connection with the neighbouring intercellular matrix.

The examination of these sections confirms Dr. Hamilton's observations that a porous antiseptic sponge, placed upon a granulating surface, will become infiltrated with new tissue; and that its own substance gradually disappears. It does not, however, seem to us at all necessary to call in the aid of Dr. Hamilton's ingenious hypothesis as to the pushing out of capillary loops, in order to explain the formation of the granulation

tissue. We very much doubt, indeed, whether the formation is, as he appears to suppose, primarily dependent on the presence of capillaries. It is possible to conceive that, from stimulation or otherwise, a proliferation of the cells of the old tissue takes place; into the new growth so formed branches from the sub-jacent capillaries may extend, by growth; from these other sproutings and continuations may arise; and in the same new tissue new capillaries may be formed, to become subsequently connected with the others. In short, the appearances observed are not inconsistent with some of the other and more generally received views.

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#### DESCRIPTION OF PLATE XVI.

Fig. 1. Capillaries (*b*) anastomosing among the fibres (*a*) of the sponge skeleton. This is near the surface of the granulation tissue. The sponge had been fourteen days in the wound.

Fig. 2. A capillary in a somewhat older growth, giving off a branch at *a* (about  $\times 400$ ).

Fig. 3. Cells of the new tissue, with processes (about 500).

Fig. 4. Giant-cells at end of a fibre.

Fig. 5. Giant-cells at ends of fibres. At *c* a distinct indentation in a fibre is seen. The smaller giant cell (*d*) has but one nucleus. The cells of the surrounding tissue are approximately of the size of *c*.

Figs. 6, 7, 8, and 9. Giant-cells more or less enveloping fibres.

Fig. 10. A large cell connected by processes with the neighbouring ones.

THE VALVULAR ACTION OF THE LARYNX. By  
T. LAUDER BRUNTON, M.D., F.R.S., and THEODORE CASH,  
M.D., *St. Bartholomew's Hospital, London.*

CLOSURE of the glottis plays a most important part in all expulsive acts, such as coughing, sneezing, vomiting or defæcation; or in those muscular actions where it is necessary to have the thorax fixed, in order to enable the muscles attached to it to act with greater advantage or greater precision. On looking at the human larynx, it not unfrequently happens that the mere act of introducing the mirror into the fauces excites movements of retching. The appearance which the larynx then presents, is that of a somewhat circular or slightly elliptical opening completely filled by three bulging segments, strongly reminding one of the appearance of the aortic valves, as seen from below in an injected aorta. On consulting several text-books on physiology we find that the mode of closure of the glottis is treated in a very cursory way. In the wonderfully complete physiology of Haller we can find no definite information, nor is there any in Todd's *Cyclopædia of Anatomy and Physiology*. In Müller's *Physiology*, translated by Dr. Baly, 2nd edition,<sup>1</sup> we find the statement that in holding the breath the air tubes are cut off from the mouth and nostrils by approximating the posterior palatine arches, and pressing the root of the tongue against the palate. In Carpenter's *Physiology*, 9th edition, and in Foster's *Physiology*, 3rd edition, we have also failed to find a definite account of the mechanism of the closure of the glottis. In Czermak's *Treatise on the Laryngoscope*,<sup>2</sup> he states that during closure of the glottis he has observed that—

“(1) The arytenoid cartilages intimately meet at their internal surfaces and processes, and they bring the edges of the vocal cords in contact; (2) the superior vocal cords approach the inferior vocal cords, so as to obliterate the ventricles of Morgagni, at the same time they also meet in the median line; (3) the epiglottis being lowered, and its cushion becoming more prominent still, it presses against the closed glottis; the contact takes place from before backwards. These three-fold occurrences in the hermetic closure of the larynx explain the

<sup>1</sup> Vol. i. page 360.

<sup>2</sup> New Sydenham Society publications, vol. xi. 1861.

resistance which the glottis successfully opposes to the pressure of the air, without a development of much force during the effort."

Czermak also gives an accurate picture of the appearance of the glottis when completely closed during effort, though a still better one is given by Grützner in Hermann's *Handbuch der Phys.* (Band 1, Theil 2, page 59). In Grützner's picture (fig. 1)

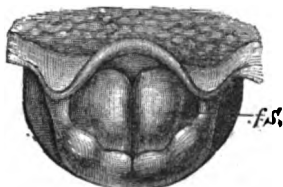


FIG. 1.—Glottis closed by the approximation of the false vocal cords (f. s.) after Grützner.

the rounded and bulging nature of the protuberances formed by the false vocal cords is very evident, and suggestive of inflation from below. Grützner, however, only remarks that the false vocal cords, or ventricular bands, are often approximated, and the cushion of the epiglottis depressed upon them, whereby a very firm closure is produced. In an inaugural thesis presented to the Edinburgh University by Dr. Wyllie in 1865, the author discussed the mode of closure of the glottis very fully, and illustrated it by numerous experiments. These showed very clearly, indeed, what an important part is played in the closure of the glottis during expiration by the false vocal cords, and that, indeed, it is chiefly if not entirely through them that the closure is usually accomplished. These experiments were published in the *Edinburgh Medical Journal*, September 1866, but as they are not referred to in many standard text-books,<sup>1</sup> it would appear that they have not received the attention they deserve. Wyllie points out that Czermak was probably deceived in regard to the second factor in the closure of the glottis.

Czermak thought that the superior vocal cords approach the inferior, so as to obliterate the ventricles of Morgagni, at the same time that they also meet in the median line.

As Wyllie points out, and as reference to the accompanying fig. 2 will show, it is impossible to say anything with certainty regarding the condition of the entrance to the ventricles of Morgagni when the glottis is closed, inasmuch as these are simply oblong orifices in the lateral walls of the larynx, and the false vocal cords completely hide them from view when they

<sup>1</sup> We must except Turner's *Introduction to Human Anatomy*, in which these experiments are referred to

are approximated. Wyllie points out that an anatomical misconception prevails regarding the nature of the vocal cords, many considering them as the free edges of membranes which are flattened above and below. They are, however, really wedge-shaped projections from the sides of the larynx, the apex of the wedge being directed downwards and attached to the laryngeal wall, its upper flat surface forming the floor of the ventricle of Morgagni, and its projecting edge forming the true vocal cord.

Their shape is as badly adapted as we can possibly imagine for retaining air in the thorax, though very well adapted for preventing air from entering it. Wyllie's experiments showed that by no adjustment could the true vocal cords completely prevent the exit of air, but that when they were simply approximated, not even being pressed together, they completely prevented its entrance. With the false vocal cords the case was just the reverse; they present no obstacle whatever to the entrance of air, but when they are approximated they completely obstruct its exit from the lungs, and the air getting behind them into the ventricles of Morgagni, inflates them, and thus the greater pressure behind them the more perfect is their apposition.

Our own investigations completely confirm those of Dr. Wyllie. He extends his experiments to the production of voice, we have restricted ourselves to the simple mechanism of the closure of the larynx during effort;

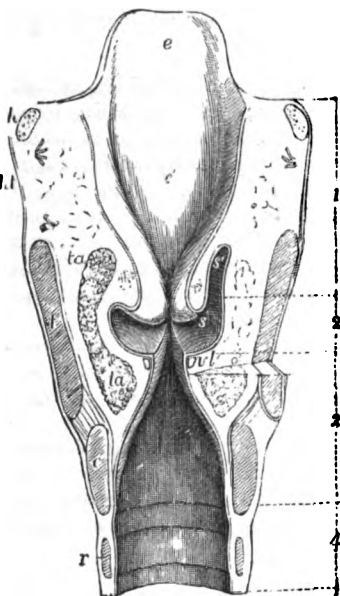


FIG. 2.—Anterior half of a transverse vertical section through the larynx near its middle. (From Allen Thomson in *Quain's Anatomy*). 1, Upper division of the laryngeal cavity; 2, central portion; 3, lower division continued into 4, part of the trachea; *e*, the free part of the epiglottis; *e'*, its cushion; *h*, the divided great cornua of the hyoid bone; *M*, thyrohyoid membrane; *t*, cut surface of the divided thyroid cartilage; *c*, that of the cricoid cartilage; *r*, first ring of the trachea; *ta*, thyro-arytenoid muscle; *vl*, thyro-arytenoid ligament in the true vocal cord covered by mucous membrane at the rima glottidis; *s*, the ventricle, above this the superior or false cords; *s'* the sacculus or pouch opened on the right side.

but instead of confining our observations to the human larynx, we have made comparative observations on the larynges of some animals.

Before entering into those in detail, it may be advisable to say a few words regarding the comparative anatomy of the larynx.

A good anatomical classification of laryngeal variations is the following, proposed by Milne-Edwards<sup>1</sup> :—

I. The aglottic type, in which the laryngeal cavity is not separated into two spaces by well-marked vocal cords.

II. The simple glottic type, in which there are well-marked vocal cords, but in which no false cords nor ventricles are present.

III. The composite type, in which the upper or anterior portion of the larynx is furnished with a second pair of cords—commonly termed false or superior cords—and which are separated from the true cords by a distinct ventricle.

IV. The cavernous type, in which the cavity of the larynx is in communication with a sinus possessing accessory pouches, of which the mouths are situated in the ventricles of the larynx, or in other parts of this organ.

In following this classification, the animals whose larynges determine their serial position will be mentioned indiscriminately, and peculiarities of the glottis briefly expressed, whilst reference to the author who is responsible for the statement will be made in a footnote.

*Class I.*—In the *pisciform mammals*, where the larynx has not the function of a vocal instrument, but only that of insuring continuity of respiratory work during deglutition, and maintaining free communication between the trachea and nasal cavities. This form of organisation is found in the Cetaceans.

The Dugong has no ventricle.

In the Dolphin<sup>2</sup> the glottis opens into the posterior nares, and has a pyramidal form. It is patent only at its summit, and leaves at either side a passage for food. The pyramidal elevation is formed by the arytenoids and the epiglottis. Probably in these, and in Cetaceans generally, there is no voice, as no true means for its production are present.

*Marsupialia.*—In the Kangaroo<sup>3</sup> there is no false cord, no ventricle, and only the faintest indication of true cords. The arytenoids are capable of considerable elevation, and permit the passage of air through a large gap between their inner surfaces.

The Kangaroo has a cavernous sinus.

It is probable that the Kangaroo is mute.

The foetal Hippopotamus has no cords but a simple longitudinal elevation, formed by the anterior extremity of the arytenoid.<sup>4</sup>

<sup>1</sup> Milne-Edwards, *Leçons sur les Physiologie et l'anatomie comparées de l'homme et des Animaux.*

<sup>2</sup> Cuvier, p. 797.

<sup>3</sup> Milne-Edwards, p. 442.

<sup>4</sup> Cuvier, p. 791.

*Class II.* is a very large one, and embraces mammals from very widely differing orders.

The Hedgehog<sup>1</sup> belongs also to this class.

In the Elephant<sup>2</sup> the arytenoids do not touch by their inner surfaces, and the true cords are placed obliquely. The false cords are indicated in position by a faint projection of the mucous membrane, and the ventricles are only formed by an excavation of the upper surfaces of the true cords. At the anterior commissure there is a transverse fold.

In the Ruminants<sup>3</sup> the arytenoids have, besides their *articular* facette, a superior angle which is curved forwards, and an inferior to which the vocal cords are attached.

The *inferior* margin of these cords is obtuse and continuous with the rest of the internal lining membrane, the *superior* margin is more or less free and trenchant—it is much more so in the Deer than in the Gazelles, and it is very indistinct in the case of the Cow and Sheep. The internal faces of the arytenoids touch, and air could only pass between the anterior margins and the epiglottis. This passage is more or less narrow according to the species. There is no superior ligament and no ventricle properly so called, neither does any cuneiform cartilage exist.

Sometimes, as in the *Antelope gutturosa*,<sup>4</sup> the thyroid is bulged outwards in the neighbourhood of the attachments of the vocal cords.

In Hares and Rabbits (*Lepus timidus* et *cuniculus*) there is an intermediary form, as the false cords are wanting, but the ventricle exists. In the *Lepus timidus*, however, Wolff denies the existence of a ventricle.<sup>5</sup>

In the Rein-Deer<sup>6</sup> there is a large subepiglottal sac, but this is not found in the Deer.

The Sloth (*Bradypus tridactylus*) has a peculiar form of vocal cord. The true cord recurves at its free margin in such a manner that with its fellow it could exert a valvular action which would seem to be sufficient to prevent the exit of air, and in the expiratory movement it vibrates with the impulse of the passing air. Cuvier<sup>7</sup> has shown that there is neither ventricle nor false cord to be found in the glottis of the *Bradypus*.

*Class III.*—Man and the greater part of Ungulate mammals are to be referred to this class, though others have the cavernous glottis which is embraced in the fourth and final division.

Here the vocal cords are not only well developed and possess a free border more or less fine, while each is capable of advancing to meet its fellow of the opposite side in the median line, but there exists above the true cords a second pair of analogous folds, less adapted to phonation, and between these projections one recognises a fossa, bilocular but not communicating with a sac or cavern. This structure is found in the Carnivora for the greater part, but this group has many varia-

<sup>1</sup> Milne-Edwards, p. 442.

<sup>2</sup> *Op. cit.*, p. 442

<sup>3</sup> Cuvier, p. 795.

<sup>4</sup> Cuvier, p. 795.

<sup>5</sup> Wolff, *Dissertatio Anatomica*, p. 19.

<sup>6</sup> Milne-Edwards, p. 442.

<sup>7</sup> *Ibid.*, p. 790.



tions amongst its members. In the Dog<sup>1</sup> (*Canis domesticus*) the larynx is very large. The true cords are well developed and broad. They are capable of being bulged to a considerable extent when air distends the ventricles, which are very deep, and ascend a considerable distance along the inner surface of the thyroid. The false cords are neither strong nor prominent. Wolff<sup>2</sup> testifies to the strength of the cords of this animal, and mentions that the ventricle is deeper at its extremities than in its middle part. *Canis lupus*.<sup>3</sup>—The ventricles of Morgagni are large and deep. In the different species of the Genus *Felis* the false cords are very prominent and well detached from the walls of the larynx. They are attached directly to the arytenoids, and at their point of juncture, under the epiglottis, they form a small vault-like attachment. In the Lion<sup>4</sup> the true cords are neither so free nor their borders so trenchant as in the Dog; they are, in fact, thick, and but slightly prominent. The superior part of the larynx is dilated. There is no ventricle according to Wolff.<sup>5</sup> In the Cat (*Felis catus*) the larynx is small, and guarded by a long upper and pointed epiglottis. Anteriorly, the false cords are widely separated; they are in structure very fine, instead of being thick as in the Lion; the true cords touch at their anterior extremities. There are no cartilages of Santorini.<sup>6</sup>

In the Tiger the arytenoids are much raised.

In the Hyæna<sup>7</sup> (*Hyæna striata*) the superior ligaments are scarcely visible, and there is but a faintly-marked ventricle.

*Plantigradu*.—In the Bear<sup>8</sup> both pairs of cords are so arranged as to raise themselves nearly to the same level by their free edges, and to direct towards the epiglottis the slit which forms the entrance to the ventricles.

Cuvier<sup>9</sup> says:—The posterior ligaments or true cords, which are thick but very distinct, and which are attached to the arytenoids, rise between the two anterior ligaments, which are attached to the cuneiforms in such a manner that the four ligaments are upon the same level, and that the ventricles of the glottis are simply two deep slits, open no longer towards the laryngeal cavity but facing the epiglottis. They bend inwards very little between the epiglottis and the thyroid. The ligaments—or rather the external anterior—are little separated from the epiglottis.

Wolff<sup>10</sup> also adopts the terms external and internal for the ligaments of the larynx as being topographically correct. He mentions, that whilst the external are inserted into the cuneiform cartilages, the internal are attached to the arytenoids. All four are inserted into the root of the epiglottis. (His examination was made upon a specimen of *Ursus arctos*).

The ventricle of the *Ursus meles* he found large and very deep.

<sup>1</sup> Milne-Edwards, p. 445.

<sup>2</sup> Wolff, *Dissertatio Anatomica*, p. 10

<sup>3</sup> *Op. cit.*, p. 10.

<sup>4</sup> Milne-Edwards, p. 445.

<sup>5</sup> *Dis. Anat.*, p. 9.

<sup>6</sup> Wolff, *Dis. Anat.*, p. 8.

<sup>7</sup> *Op. cit.*, p. 9.

<sup>8</sup> Milne-Edwards, p. 446.

<sup>9</sup> *Anat. Comp.*, Tom. viii., p. 787.

<sup>10</sup> *Dis. Anat.*, p. 12.

The *Erinaceus Europæus*<sup>1</sup> has only small superior cords, though the ventricles are deep and sacculated.

The Coati<sup>2</sup> has a somewhat similar arrangement to the Bear, but the Badger<sup>3</sup> has ligaments of the usual position, the anterior with a sharp margin, the posterior, however, being obtuse. The ventricle is open and leads into a sac. The sound which this animal emits is probably produced by the friction or impaction which the air suffers against the posterior border of the anterior cords and its division into these diverticula.

The Civet<sup>4</sup> has a glottis like that of the Cat.

It results from the structure of the larynx in the genus of the cat tribe, that it is chiefly the anterior ligaments which must perform the functions of vocal cords. Their union towards the epiglottis forming a little vault against which the air must strike with force, favours this view.<sup>5</sup>

Movements of the false cords only become apparent in great expiration and inspiration. In phonation they do not advance in the manner of a platform above the true cords, but they depress and apply themselves on the basal portion of these so as almost entirely to efface the entry of the ventricle of the larynx, and to limit the vibrations of the true cords to a certain length.<sup>6</sup> As the voice rises, these membranous folds cover progressively from without inwards the vocal cords.<sup>7</sup>

*Pinnigrada*.—The Seal<sup>8</sup> has an obtuse vocal cord which is but slightly free. The anterior ligament blends with the base of the epiglottis. The ventricle is superficial.

The Marmot has a very sharp margin of the anterior ligament, more so, in fact, than that of the posterior.<sup>9</sup>

*Class IV.* may be regarded as containing animals which would belong to the third class, had they not some "cavernous" character superadded, and also animals of a less perfect type.

The Llama has false and true cords, a ventricle, and a common larynx, and therefore with the Camel (*Camelus bactrianus*) forms an exception to the usual Ruminant type. (The latter animal has a trachea 3 feet in length, but very narrow; its larynx is small and its voice proportionately weak.)<sup>10</sup>

In Solipedes<sup>11</sup> the vocal cords are narrow and situated deeply. There are no false cords, and no ventricle properly speaking, but a hole pierced in the lateral wall above the true cords conducts into a large oblong sinus, hidden between the wall and the thyroid, and covered to a great extent by the thyro-arytenoid muscles, which should be able to compress it.

This opening is large in the Horse,<sup>12</sup> but the cavity is not very deep.

<sup>1</sup> *Op. cit.*, p. 15.

<sup>2</sup> Cuvier, p. 788.

<sup>3</sup> *Ibid.*

<sup>4</sup> *Ibid.*

<sup>5</sup> *Ibid.*, p. 787.

<sup>6</sup> Milne-Edwards, p. 512.

<sup>7</sup> *Ibid.* p. 525.

<sup>8</sup> *Ibid.*, p. 788.

<sup>9</sup> Cuvier, p. 789.

<sup>10</sup> *Dis. Anat.*, p. 23.

<sup>11</sup> Cuvier, p. 793.

<sup>12</sup> *Ibid.*, p. 793.

There<sup>1</sup> is also in this animal a triangular membrane situated in the angle of the thyroid. This easily vibrates as it rests upon the moving cords beneath it. The commencement of the "hinny" is due to repeated shocks of expired air upon this membrane.

In the Ass<sup>2</sup> two sacs are situated above the attachment of the cords. A tendinous membrane is also present. There is a great deepening of the thyroid.

Cuvier<sup>3</sup> considers the larynx of the Mule more allied to that of the mare, and speaks of Herrissant's observations as inaccurate, and as regards the triangular membrane of the horse's and ass's larynx, exaggerated.

The Rhinoceros<sup>4</sup> has well-marked vocal cords and deep ventricles, before each of which is a nearly vertical opening, and it is at the bottom of this excavation that the anterior ligaments are attached.

In the Pig<sup>5</sup> the true cords are free and sharp. The superior ligament is large and its margin rounded. The ventricle is shallow, and from this opens an oblong sinus which rises up between the mucous membrane and the thyroid, and is the size of the end of the little finger. . . . . (It is to be observed that in many animals of this class, i.e., quick runners,<sup>6</sup> the two cartilages are arranged in such a manner as not to coaptate completely, and so allow a space to remain free between them when they are touching by their summits. It results from this, that by the approximation of the true cords the glottis is not closed, and that there remains always a passage for the air behind the membranous part. Mr. Mandl thinks that this is peculiar to the species which run rapidly.)

In the Howling Ape<sup>7</sup> (*Mycetes*) the hyoid is enormously developed in form of a bell to lodge the air sacs peculiar to this animal. There are also air sacs in the pharynx.

In *Simia sabæa*<sup>8</sup> at the root of the epiglottis, and above the ligaments, there is a transverse opening which leads to a membranous sac situated between the thyroid cartilage and the hyoid bone. In the walls of the sac the fibres are partly tendinous and partly muscular.

In *Simia parnisco*<sup>9</sup> there is a sac between the cricoid and trachea. The trachea has continuous rings in some Simians.

If the view that the function of the false cords or ventricular bands is to close the glottis during effort, and thus to fix the thorax, we should expect them to be very strongly developed in those animals whose habits render such fixation likely to be serviceable; on the other hand, we should expect them to be absent in those animals where fixation of the thorax would be of little or no service; and this seems to be actually the case.

<sup>1</sup> Herrissant, *Récherches sur les Organs de la Voix*, p. 282.

<sup>2</sup> *Op. cit.*, p. 285.

<sup>4</sup> *Op. cit.*, p. 791.

<sup>6</sup> Milne-Edwards, p. 455.

<sup>8</sup> Wolff, p. 1.

<sup>3</sup> Cuvier, p. 793.

<sup>5</sup> Cuvier, p. 791.

<sup>7</sup> *Op. cit.*, p. 448.

<sup>9</sup> *Ibid.*, p. 1.

In animals whose motions are chiefly those of running, we find the ventricular bands absent, or slightly developed. But in animals where the anterior extremities are used for striking, hugging, or climbing, the vocal cords are strongly developed. We might at first expect also, that in cases where the anterior extremities were employed for the purpose of prehension, we would also find the ventricular bands developed. But this is not always the case.

When engaged in any very delicate work where the least oscillation of the hand might be injurious, we often hold our breath, but for ordinary prehensile actions we do not close the glottis, unless considerable effort is required at the same time. In marsupials such as the kangaroo, the anterior extremities are used for holding food and conveying it to the mouth. We might therefore expect that the false vocal cords would be strongly developed. But the fore limbs are small and weak, and very slight muscular effort is employed in the movements just mentioned. The fact that in these animals the ventricular bands are absent, is, therefore, very much what might have been expected. In the solipedes they are also absent. In the pig they are rounded; and there is a shallow ventricle in the hedgehog. They are present, but small, in the llama, and in the camel they are fairly well marked. In the dog the true cords are well developed and broad; the ventricular bands are not strong or prominent, but the ventricle of Morgagni is deep. In the wolf the ventricle is also deep and large. In the lion and tiger the ventricular bands are prominent and well detached from the walls of the larynx. In the cat they are not large; and they are very fine instead of being thick as in the lion. In the three-toed sloth they are well developed. In the bear, in which the closure of the glottis would require to be specially strong, from its habit of climbing trees and destroying its enemies by hugging, the arrangement of the ventricular bands is very remarkable; the vocal cords are capable of being raised until they and the ventricular bands are nearly at the same level, and the opening between them is directed towards the epiglottis from which the false cords are but little separated. During the closure of the glottis the cushion of the epiglottis will, therefore, to a considerable extent, be directed against the opening of the

ventricles, and the glottis will, we should think, be closed with very great firmness.

We have, however, not seen any specimen of the larynx of the bear, and these considerations are drawn only from the description which we have read.

In the howling monkey the ventricular bands are well developed.

Our own experiments were made upon the fresh larynx of the sheep, of the dog, of the cat, of the ape, and of man.

The experiments were made by fixing a T-cannula in the trachea below the larynx. The lower arm was connected with a bellows, and the side branch with a water or mercurial manometer; the arytenoid cartilages and the vocal cords were then approximated, as well as the ventricular bands when these were present. The strength of current which these structures could resist in various positions, and during inspiration and expiration, was estimated by the height at which the water or mercury stood in the manometer. A curved needle was passed through the bases of the arytenoids which were then coaptated by means of a figure-of-eight ligature. In some experiments in which the larynx possessed cords of such dimensions as to admit of it, needles were passed through the thyroid cartilage, one on either side of the middle line, and just external to the anterior attachment of the false cords, and the points were pushed backwards inside the edge of the false cord towards the arytenoid cartilage. In this way, approximation of the cords could be easily produced by movement (separation) of the eye end of the needle. In smaller larynges, however, approximation was assisted by seizing the coaptated edges of the cords with a pair of fine pointed curved forceps. Lateral pressure was exercised by means of a weighted scale pan which was connected with a movable concave surface of wood placed externally over the line of attachment of the cord to the wall of the larynx.

This was aided or substituted by manual pressure, and manipulation was also resorted to in pushing the base of the tongue with the epiglottis backwards over the larynx.

In the sheep the ventricular bands are absent. The following figures represent the result of these experiments :—

On blowing upwards through the glottis, the true vocal cords being closed (no false cords).

				RESISTANCE.	
				In millimetres of mercury.	In inches of water.
Experiment	1	...	...	4	2
"	2	...	...	5	2.5
"	3	..	...	8	4.25
Average about				5.5	3

On sucking air downwards through the glottis, the resistance rose to heights varying from 54 to 140 millimetres of mercury, or from about 28 to 72½ inches of water.

In the cat the resistance to the exit of air from the larynx presented by the true vocal cords alone is very small, generally about 6 mm. of mercury.

When their approximation is aided by a lateral pressure of 100 grammes, a resistance of 16 mm of mercury may be reached.

The ventricular bands in the cat are thin and easily coapted, but they are not calculated to resist much pressure unless they are supported: such support is afforded by the epiglottis, if it is slightly pressed backwards, and gentle lateral pressure is made so as to coaptate the cords.

If firm pressure is applied in this way the resistance presented to the exit of air by the ventricular bands is very considerable, and they may not yield even under pressure of from 24 to 40 mm. of mercury. To reach the latter figure, however, considerable support from the base of the epiglottis and from the lateral aspects of the larynx is required. When the true cords are approximated, as well as the ventricular bands in the cat, the epiglottis being at the same time slightly depressed and gentle lateral pressure exerted, they easily resist a pressure of 30 mm. of mercury, or more.

The resistance to the ingress of air afforded by the true cords alone, in the cat is very considerable; when a lateral pressure of 20 grammes is exerted so as to bring them together they easily resist a suction power of 50 or 60 mm. of mercury, and when the lateral pressure is increased they will resist considerably greater suction than this.

The ventricular bands when ordinarily approximated will not resist more than two or three mm. of mercury.

In such experiments as these there is not a little difficulty in approximating the vocal cords so as to imitate their closure during life. The lateral pressure exerted, the position of the epiglottis in regard to the cords, the freshness of the larynx, and many other circumstances modify the results obtained to a very considerable extent. By way of example we may mention that in eight measurements of the resistance offered by the approximation of true and false cords in the cat, at the same time, variations from 30 to 60 mm. were registered. The former figure is, however, the more exact, though, if there be a slight increase of lateral pressure, and the epiglottis be pushed gently backwards over the glottis, a pressure of 40 and 60 mm. may be obtained.

The following figures in mm. of mercury may be taken as representative of the results:—

BLOWING.			SUCTION.		
True and False.	True alone.	False alone.	True and False.	True alone.	False alone.
30-50	4-10	24-40	60-120	60-120	2-10

It appears from these figures that closure of the superior cords did not increase the powerful resistance offered by the inferior to the ingress of air as represented by suction. A quick powerful suction (*i.e.*, inspiration) movement closes the true cords, but this does not affect the false cords in the same manner, nor does the negative pressure in the trachea extend to them. The larynx again tends to pass downwards, and the "telescoping" due on the one hand to the elevation of the larynx, and on the other to the pressing backwards of the root of the tongue and of the epiglottis with its supporting pad, which occurs in efforts to vomit, is here entirely absent.

We could, however, imagine that the ventricle becoming distended in expiration after narrowing of the larynx by muscular action and approximation of the false cords, the inferior might themselves be closed by the pressure from above, and that thus a slight measure of support might be afforded.

The false cords in the dog are inconspicuous and weak in comparison with the broad well-developed true cords.

They offer almost no resistance to the ingress of air: 4 mm. of mercury being the utmost, and this amount is only reached when sticky mucus on the bands adds to the resistance.

The true cords, either alone or with the false cords, resist the ingress of air with a force of 80 to 180 mm. of mercury.

The true cords alone offer a resistance of only about 9 mm. to the exit of air.

The ventricular bands alone offer 18.5 mm., and this may be increased to 60 mm. if the glosso-epiglottidean sulcus be thoroughly distended; and the true and false together, 21 mm.

The relatively high pressure of the true and false cords together was accompanied by a bulging upwards of the glosso-epiglottidean sulcus. It is possible that the inflation of the ventricle tends also to push the true cords together, and that thus they aid to a slight extent in closing the glottis during expiration.

The larynges examined were those of small dogs.

In the ape, when the ventricular bands alone are approximated, inflation of the ventricles is well marked at the bases of the bands. It is distinctly seen in this animal that powerful expulsion of air, and especially sudden expulsion, tends to close the ventricular bands, and this strongly confirms the views already expressed in regard to the function of these structures.

A sudden inspiratory effort when both true and false cords, or when the true cords alone, are approximated, is seen to cause the lateral fossæ to sink considerably downwards, and the resistance to the ingress of air which they afford is very great: being equal to 75 mm. of mercury and more.

When the ventricular bands alone are approximated, their power of resistance is very small, unless they are firmly coaptated, when they present considerable resistance to the ingress of air. But it is very improbable that in the living larynx they can be pressed together in this way; and their functions, therefore, are evidently very different from that of the true cords, in which the simple occurrence of a rapid inspiratory movement, when the true cords are approximated, effectually closes the glottis.



## OLD WORLD APE (Small Specimen).

PRESSURE.			SUCTION.		
True closed and False.	True closed.	False closed.	True closed and False.	True closed.	False closed.
80 mm.	2-5m.	24m.	70-140	70-140	2-4

Only two specimens of the human larynx were examined.

The ventricular bands alone afforded a resistance of 30-0 mm. when gentle lateral pressure was employed. Whilst blowing air from below, a slight telescoping, by pressing the base of the tongue and the epiglottis backwards, being permitted, a bulging upwards of the hyoid fossa and the root of the ventricle underneath the attachment of the ventricular bands could be distinctly seen. When firm lateral pressure was associated with this movement a much higher pressure was resisted. The ventricular bands alone, when not forcibly held together, presented no resistance whatever to inspiration—in fact, they separated even when the true cords were in contact, and resisting a powerful suction.

The true cords alone resist the ingress of air quite as much as when the ventricular bands also are approximated. The resistance is very great, and sometimes reaches 140 mm. of mercury, and even more—in fact, suction of greater power than we could employ failed to separate them, but rather tended to increase their resistance.

Closure of the glottis is so important a factor in the act of vomiting, that we must now consider how far the development of the false vocal cords in different classes of animals is associated with the easy and perfect performance of the act.

We find that in *Ruminants* generally true vomiting is either difficult or impossible. The same is the case with the *Solipedes* and the *Rodentia*, while in the cat and dog it is performed most effectually. There is no doubt, in the act of vomiting, another factor than that of simple increase in intra-thoracic, or, more accurately, intra-tracheal pressure, for however greatly this pressure be increased, as in coughing, defæcation, or parturition, vomiting does not occur unless the cardiac extremity of the œsophagus be dilated. One explanation of the difficulty with which vomiting occurs in horses, for example, is that the

œsophagus passes a considerable way below the pillars of the diaphragm, and that thus the fibres which radiate from it on to the stomach tend to exert rather a longitudinal than a lateral action, and to pull the œsophagus down, or the stomach up, rather than to dilate the orifice. This explanation may, to a considerable extent, be correct, but we think that the other factor, viz., the want of true vocal cords, and the consequent difficulty of greatly increasing the intra-thoracic pressure, is also a factor which ought not to be entirely disregarded. We have made some experiments on the intra-thoracic pressure in vomiting: these were performed by narcotizing an animal with ether, passing the arms of a T-shaped cannula upwards and down-



CHART A.—Retching movements of cat, showing sustained intra-tracheal pressure of 12·5 mm. mercury.

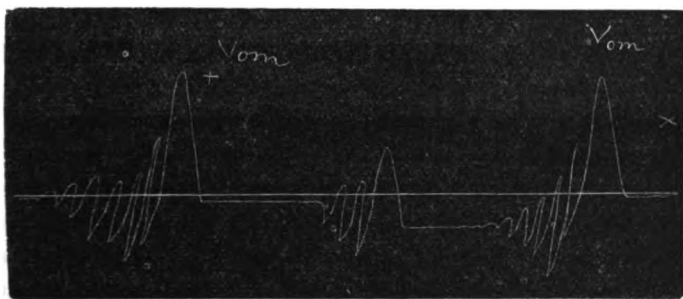


CHART B.—Vomiting movements of same, showing resistance to intra-tracheal pressure yielding at  $\pm$  to 31 mm. mercury.

wards into the trachea, the cross-limb of which was connected with a manometer. Sulphate of zinc was then injected into the stomach, and the action of the anæsthetic was diminished to the same point as in surgical operations where vomiting occurs before the return of consciousness. The results will be seen from the portions of the curves which were obtained and which we here append.

The middle curve was merely of retching.

The secondary waves arise from mercurial oscillation.

By the same kind of experiment it was proved that vomiting was still possible when the projecting arm of the T-cannula was unconnected with the manometer, and left open, but in this case vomiting occurred with much greater difficulty than when the resistance of the mercurial column was brought to bear in closing the trachea, and thus permitting the cords to resist the pressure favourable to this action.

### CONCLUSIONS.

Our experiments completely confirm those of Dr. Wyllie. The ingress of air into the glottis is prevented by approximation of the true vocal cords, but these have very little power to prevent its egress. The false cords or ventricular bands, on the contrary, have very little power to prevent the ingress of air into the lungs; but when the edges are brought together they act as valves and offer great resistance to the egress of air: they are, therefore, to be regarded as the chief factors in the closure of the glottis during exertion.

Although our data are insufficient to enable us to speak with certainty, so far as they go, they appear to show that, in such animals as do not require to have the thorax fixed, ventricular bands are rudimentary or absent: that in those animals where fixation of the thorax is advantageous for giving greater precision or force to the movements of the anterior limbs in striking, climbing, or hugging, the ventricular bands are well developed. This condition is seen in the cat, lion, sloth, bear, ape, and man.

Though the power of any species of animal to vomit is not entirely dependent on the presence of the false cords, yet when present, their action in closing the glottis is an important factor in the act of vomiting.

See, for description of larynges referred to, the following authors, from the works of some of whom we have translated largely—

- Milne-Edwards, *Leçons sur la Physiologie et l'Anatomie Comparée de l'Homme et des Animaux*, xii. pp. 422 et seq.  
 Cuvier, *Anatomie Comparée*, viii. pp. 772 et seq.  
 Wolff, *Dissertatio Anatomica de Organo Vocis Mammalium*.  
 Herrisant, *Récherches sur les Organes de la Voix*.  
*Cyclopædia of Anat. and Phys.*, vol. iv. pt. ii., "Voice."  
 Turner, "*Balanoptera Sibbaldii*," *Trans. Roy. Soc. Ed.*, xxvi.  
 Watson & Young, "Anatomy of Northern Beluga," *Trans. Roy. Soc. Ed.*, vol. xxix.

ORIGIN OF THE INTERNAL CIRCUMFLEX FROM  
THE DEEP EPIGASTRIC ARTERY. By ARTHUR  
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Edinburgh.*

MY attention has been directed this winter to two instances of the somewhat rare condition in which the internal circumflex artery of the thigh takes origin from, or in common with, the deep epigastric artery.

These examples occurred in the right thighs of two female subjects dissected in the University rooms.

Special interest attaches to this abnormal condition, as the irregular arteries have a more or less intimate relation to the femoral canal; and hence might be endangered in the operation for the relief of a femoral hernia.

Under these circumstances I shall first proceed to describe the origin, course, and relations of the irregularly-placed arteries.

In the first of the two examples I found it springing from a trunk common to it and the deep epigastric. This trunk, which took origin from the external iliac, about one-third of an inch above Poupart's ligament, passed transversely inwards for about one quarter of an inch, being in front of, and across, the external iliac vein. It then divided into the deep epigastric, and an artery which took the place of the internal circumflex. The latter, curving inwards and downwards, pierced the femoral sheath just below Poupart's ligament, in this part of its course lying internal to the femoral vein, and crossing the crural canal.

Resting on the pectineus muscle, it was bound down to the front of that muscle by the fascia covering it. In this position, where it lay fully one inch internal to the common femoral, it gave off the deep external pudic branch.

On this fact I lay considerable stress, as it would tend to fix the artery, and so prevent a femoral hernia passing to its inner side or pushing it downwards before it.

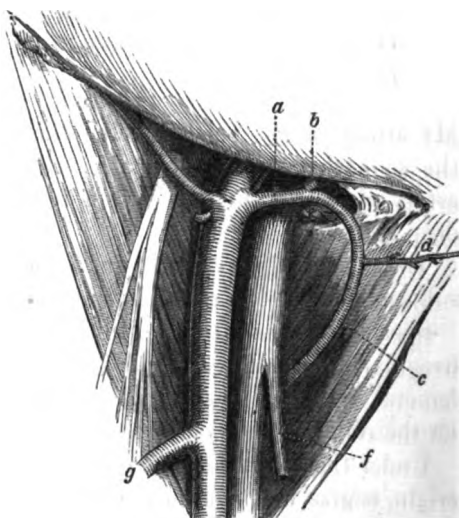
The artery then curved downwards and outwards, lying

behind the femoral vein at its junction with the internal saphenous, and passing deeply between the psoas and pectineus muscles.

In the second instance a common trunk was seen taking

#### EXPLANATION OF FIGURE

- a. Common trunk of deep epigastric and internal circumflex arteries.
- b. Deep epigastric artery.
- c. Internal circumflex artery.
- d. Deep external pudic artery.
- e. Gimbernat's ligament.
- f. Internal saphenous vein.
- g. External circumflex artery.



origin from the external iliac about a quarter of an inch above Poupart's ligament, and passing inwards across the vein. This portion of the trunk had a curved course, the convexity of the curve being upwards.

At a point corresponding to the septum, between the compartment for the vein and the crural canal, the artery pierced the sheath and split into the deep epigastric and internal circumflex; the latter coursed directly downwards, being in contact with and to the inner side of, the vein, and in front of the partition between the femoral canal and compartment for the vein.

About an inch and a quarter below Poupart's ligament it disappeared behind the vein, to pass deeply between the psoas and pectineus. In its course downwards it *did not* give off the deep external pudic; this branch being observed to take origin from the superficial femoral, about two and a half inches below Poupart's ligament.

From what has been stated above, it will be seen that the two arteries differ in two very important details.

The first specimen was found lying upon the pectineus, being

bound down to the surface of that muscle by the strong fascia covering it, and it was also observed to give off the deep external pudic.

The artery in the second specimen did not reach the anterior surface of the pectineus until it lay at a lower level than the femoral canal, and it was not fixed in its position by any branch.

Thus it is clear that whereas the former passed at first across, and then to the inner side of the crural canal, the latter lay altogether external to the canal.

By these relations it would thus appear that, had a femoral hernia existed in the former condition, the artery would in all probability have surrounded the neck of the sac, and would inevitably have been injured, had its presence not been ascertained during the progress of an operation for the relief of strangulation. That this latter is quite possible is proved by the case recorded by the late Professor Spence in his lectures and published text-book, in which he was enabled to ascertain the presence of an irregular obturator, and apply a ligature round the artery before dividing it. Had a hernia, on the other hand, existed in the second example which I quote, the artery would have lain to the outer side of the sac and not been endangered.

Here it may be well to draw attention to the similarity between the cases of irregular obturators springing from the deep epigastric, and such examples as these herein mentioned. Just as we find those irregular obturators lying at times external to, at other times encircling, the neck of the sac, so, were a hernia to occur with an irregular internal circumflex artery, the artery might either lie to the *iliac* or to the *pubic* side of the neck of the sac, and I hold that there is reason for supposing that the latter (*i.e.*, pubic) variety is co-existent with the origin of the deep external pudic from the irregular internal circumflex.

In other respects there is also some similarity between the two conditions. Thus it will be seen that the origin and course of an irregular circumflex such as I have described crossing the crural canal, are almost identical with the origin and course of an irregular obturator surrounding the neck of the sac; that is to say, they correspond in position until they reach Gimbernat's

ligament. Here the latter is in relation to the upper and inner surface of the ligament, whereas in the former case the artery is in relation to its inferior and external surface; hence its presence and relation to Gimbernat's ligament would be much more readily recognised, and the application of ligatures to it much simpler.

Several instances of this irregularity have been recorded.

Burns<sup>1</sup> mentions two cases in which the internal circumflex would be exposed to great danger in the operation for femoral hernia. The one arising from the external iliac he describes as running "along the front of the lymphatic sheath"; the other sprang from the femoral, a little below the crural arch, and "traversed the front of the common sheath of the great vein and also of the lymphatics."

Dr. John Reid<sup>2</sup> also cites a case of the internal circumflex artery taking origin from the deep epigastric, but observes "that the artery would in all probability have lain on the iliac side of the neck of the sac."

Michelet<sup>3</sup> records an internal circumflex iliac coming off in common with the deep epigastric, and expresses his opinion that had a hernia descended it would have pushed the artery to the pubic side of the neck of the sac.

Quain illustrates two instances of irregular origin of the internal circumflex from the epigastric. In fig. 3, plate 74, the artery curves over the femoral vein. In fig. 4 of the same plate the internal circumflex rises in common with the obturator and deep epigastric, and passing round the femoral vein, reaches its destination. In fig. 1 (same plate) we also see an internal circumflex artery taking origin in common with the deep epigastric, and here it passes backwards between the femoral artery and vein.

He only found this arrangement, viz., springing from epigastric, in one of 391 cases.

But he seems to be of opinion that in none of these conditions would the artery be endangered in a case of femoral hernia.

<sup>1</sup> "Observations on parts concerned on Crural Hernia," *Edin. Med. and Surg. Jour.*, vol. ii.

<sup>2</sup> "Anatomical Observations," *Edin. Med. and Surg. Jour.*, vol. xlv. p. 69.

<sup>3</sup> Thèse. inaug. 1837, quoted from Dr. Reid's paper.

Wood<sup>1</sup> furnishes an instance of high origin of this artery from the common femoral, in which it crossed the femoral vein within the sheath, and then lay to the outer side of and below the lymphatic compartment.

In another subject (female) he found the deep epigastric, internal circumflex, and obturator branches arising by a common trunk from the common femoral just at its commencement. He describes them as follows:—

“This (the common trunk) crosses in front of the femoral vein just below Poupart’s ligament. The abnormal circumflex and obturator will be seen both to hug closely the femoral vein on its inner side, and would necessarily be placed on the outer side of a crural rupture.”

This case will be seen to differ from that which I have described, as here the artery takes origin from the common femoral, whilst that mentioned by me took origin from the external iliac, and passed inwards.

From the two cases I have recorded, taken in conjunction with those instances which I have quoted, it appears that when an irregular internal circumflex artery takes origin from or in common with the deep epigastric artery, it may have one or other of the following relations to the femoral canal:—

1. It may cross the crural canal, and the artery would in this position surround the neck of the sac were a hernia to exist. This variety I consider only exists when the artery, as it lies in front of the pectineus, is fixed in position both by the fascia covering that muscle and by the deep external pudic artery there taking origin from it, these two circumstances preventing the displacement of the artery downwards or outwards.

2. The artery may lie either in front of the lymphatic sheath or altogether external to it; here the artery either passes deeply to the inner side of the vein below the level of the crural canal, or else dips backward between the artery and vein. In this condition the artery is freely movable, and would readily be pushed aside by a hernia being placed to the outer and posterior aspect of the sac.

<sup>1</sup> Wood on *Rupture*, p. 195.



CERVICAL RIBS, AND THE SO-CALLED BICIPITAL RIBS IN MAN, IN RELATION TO CORRESPONDING STRUCTURES IN THE CETACEA. By PROFESSOR WM. TURNER, M.B., F.R.S.

IN the earlier volumes of this *Journal* I gave an account of some specimens of cervical ribs,<sup>1</sup> and of specimens of ribs with two heads<sup>2</sup> which I had examined. As during the past year two subjects were brought into my dissecting rooms in which cervical ribs were present, and other two in which certain of the ribs were cleft at their vertebral ends, I have been led to look anew into these peculiarities of structure as they present themselves in Man and in the Cetacea.

CERVICAL RIBS.

*Case A.*—Adult man with a pair of cervical ribs. On the *left* side the cervical rib had a head, neck, tubercle, and rudimentary shaft. The head articulated by a movable joint, which had a strong costo-central ligament with the upper side of the body of the 7th cervical vertebra, and by its tubercle with the transverse process of the same vertebra. The shaft was a curved slender bar of bone 1·4 inch long. It terminated anteriorly in a point, from which a fibrous band passed forward to join a cartilage 0·6 inch long, which fused with the cartilage of the 1st thoracic rib immediately below the right sternoclavicular joint. The scalenus anticus was attached partly to the fibrous band and partly to the bony shaft of the cervical rib close to its termination. The subclavian artery and the lowest cord of the brachial plexus rested in a groove on the upper surface of the rudimentary shaft immediately behind the scalenus anticus. This artery was elevated above its usual position, but the anterior end of the cervical rib did not make any special projection in this region. The scalenus medius was attached partly to the rudimentary shaft behind the subclavian artery, and partly to the 1st thoracic rib. The tendon of the subclavius arose partly from the sternal cartilage of the cervical rib, and partly from that of the 1st thoracic rib. The space between the different elements of the cervical costal arch and the 1st thoracic rib was occupied by two planes of muscle, an internal and an external intercostal muscle. The upper border of the external intercostal ended in a line with the anterior end of the bony shaft of the cervical rib.

<sup>1</sup> "On Supernumerary Cervical Ribs," vol. iv., Nov. 1869.

<sup>2</sup> "The so-called Bicipital Ribs in Whales and in Man," vol. v., May 1871.

but the internal intercostal was prolonged inwards close up to the sternum. The vertebral artery ascended in front of the neck of the cervical rib to enter the foramen at the root of the 6th cervical transverse process. The art. profunda cervicis passed back between the necks of the cervical and 1st thoracic ribs.

On the *right* side the cervical rib had a head, neck, and tubercle, with corresponding attachments to those seen on the opposite side. The shaft was slender, and extended only 0·9 inch beyond the tubercle. It ended externally in a point, from which a fibrous band passed obliquely forwards to be attached to the upper surface of the 1st thoracic rib behind the insertion of the scalenus anticus, which muscle was inserted into that rib only, and not into the cervical rib. The right cervical rib had therefore no connection with the manubrium. The subclavian artery and lowest cord of the brachial plexus rested on the end of the bony shaft of the cervical rib, so that they were both elevated above their usual position. The scalenus medius was attached partly to the rudimentary shaft, but chiefly to the 1st thoracic rib. The interval between the rudimentary cervical rib and the 1st thoracic rib was occupied by a pair of intercostal muscles, the internal of which passed as far as the fibrous band which connected the cervical rib to the 1st thoracic. The vertebral and deep cervical arteries had similar relations to the cervical rib as on the left side. There were twelve pairs of thoracic ribs. The left 12th rib was 1·4 inch long, the right 2·8 inches.

In this subject the right subclavian artery arose as the last branch of the transverse part of the arch of a left aorta, and passed from left to right behind both trachea and oesophagus and in front of the spine, in order to reach the root of the neck on the right side. This variation in the origin of the right subclavian is not uncommon. Another case occurred in the practical rooms during the same session, and one in the previous session, whilst many cases have on previous occasions come under my notice.

*Case B.*—Old woman with a pair of cervical ribs. On the right side the rib had a head, neck, tubercle, and rudimentary shaft. The head articulated by a movable joint with the upper part of the side of the body of the 7th cervical vertebra, and by its tubercle with the transverse process of the same vertebra. The shaft was 1·4 inch long and 0·3 inch broad near the tubercle, but then became styloid in shape, and its pointed anterior end was connected by a strong fibrous band to the 1st thoracic rib, where the cartilage and bone became continuous with each other. The 1st rib was broadened out at this spot to 1·4 inch. Its costal cartilage was attached in the usual way to the side of the manubrium. A pair of intercostal muscles occupied the interval between the cervical costal arch and that of the 1st thoracic rib. The scalenus anticus was attached to the fibrous band. The subclavian artery rested in a groove on the styloid part of the cervical rib. The scalenus medius and posticus were not well differentiated from each other, but a part of their common tendon was inserted into the shaft of the cervical rib behind the subclavian artery, whilst

another and larger part descended to the upper border of the 1st thoracic rib. The lowest cord of the brachial plexus rested in a groove on the posterior part of the shaft of the cervical rib. The subclavian vein was in front of the tendon of the anticus, and rested on the fibrous band.

On the left side the cervical rib had similar articulations to the spine as on the right side. The bony shaft was 1·2 inch long, and was not so attenuated as the right. Its anterior end was pointed, and joined a fibrous band, which ended in a costal cartilage that was parallel to and immediately above the cartilage of the 1st thoracic rib, with which it blended prior to the attachment of that cartilage to the side of the manubrium. The greatest breadth of the 1st thoracic rib was 0·9 inch. The scalenus anticus was inserted into the fibrous band, and the medius into the bony shaft of the cervical rib and the 1st thoracic rib. A pair of intercostal muscles occupied the interval between the cervical and 1st thoracic costal arches. The lower cord of the brachial plexus rested in a groove on the shaft of the cervical rib; both the subclavian artery and vein rested on the fibrous band—the one behind, the other in front of the scalenus anticus.

The vertebral artery on each side entered the foramen in the root of the 6th cervical transverse process, and the deep cervical artery passed backwards between the neck of the cervical and that of the 1st thoracic rib.

The spine of this old woman was greatly curved; the ribs were distorted, the pelvis was contracted, and the bones softened from *mollities ossium*. There were twelve pairs of thoracic ribs. The twelfth right was  $1\frac{1}{2}$  inch long; the twelfth left,  $2\frac{1}{4}$  inches.

These two cases of cervical ribs illustrate the higher grades of development of a costal arch belonging to the 7th cervical vertebra. For, although in neither is the arch complete in bone and cartilage, yet on the left side of A and on the right side of B the rudimentary bony shaft is continued into a costal cartilage by an intermediate fibrous band. On the opposite side of each specimen the costal cartilage is not so complete, for the fibrous band ended in the shaft of the 1st thoracic rib, although in B this junction took place close up to the costal cartilage. Had these specimens not been examined in the recent state with the soft parts in position, but after maceration had destroyed all but the bony structure, then they would have appeared as if the rudimentary bony shaft of the cervical rib had terminated in a free-pointed end. It is not unlikely that in various of the cases described by different anatomists, from the examination of the skeleton only, as cervical ribs terminating in a free end, the rib may have been prolonged forwards through a fibrous band similar

to what I saw in my specimens, either to the shaft of the 1st thoracic rib or even to a costal cartilage. This was probably the case in the specimen which has just been described (fig. 2), by Dr. F. J. Shepherd of Montreal in a skeleton in the Museum of the M'Gill University.<sup>1</sup> It is unnecessary that I should comment further on the varieties and mode of origin of cervical ribs, as these have been elsewhere amply discussed by Hunauld, Knox, Luschka, Gruber, Struthers, and myself. I may, however, take this opportunity of re-directing attention, as indeed Dr. Knox had previously done, to M. Hunauld's essay,<sup>2</sup> in which the origin of the anterior division of the transverse process of the 7th cervical vertebra from a distinct centre of ossification is both described and figured, and it is stated that when this process increases in size and does not unite with the vertebra, then a cervical rib is formed. It is to M. Hunauld, therefore, and not to his countryman M. Béclard, that the merit of having first made this important observation should be ascribed.

#### BICIPITAL RIBS IN MAN.

This anatomical peculiarity is not due to a bifurcation of the shaft of a single rib at its vertebral end into two heads, but to the fusion of what ought to have been the shafts of two distinct ribs into a common body, and it invariably occurs at the apex of the thorax. Although I have long been familiar with specimens of this kind in the Anatomical Museum of the University of Edinburgh, the cases which I am now about to describe are the first that I have seen in the subject itself in the course of nearly thirty years' experience as a teacher of anatomy, and it is remarkable that they should both have occurred within a few months of each other.

*Case 1.*—Adult male. The vertebral formula was normal. No special anatomical arrangement in the cervical or lumbar vertebræ had to be noted.

On the *right* side the head of the 1st rib articulated with the lower margin of the side of the body of the 7th cervical vertebra with

<sup>1</sup> *American Journal of the Medical Sciences*, January 1888. I am indebted to Dr. Shepherd for a separate copy of his essay on "Cervical Ribs."

<sup>2</sup> *Mémoires de l'Académie Royale des Paris*, 1740, p. 525, 12mo, Amsterdam, 1744.

the 1st dorsal vertebra and the intermediate fibro-cartilage. Its tubercle articulated with the transverse process of the 1st dorsal, and its shaft fused with that of the 2nd rib immediately external to the tubercle, and 0·8 inch from the side of the body of the 1st dorsal. The head and tubercle of the 2nd rib had the usual articulations.

A groove on the outer surface of the body common to the 1st and 2nd ribs marked their line of fusion. This common body attained a maximum width of 1·2 inch, and the direction of its surface was not horizontal but oblique. On the outer surface was a roughness for the origin of the serratus magnus. It bifurcated anteriorly  $3\frac{1}{2}$  inch in front of the tubercles of the ribs. The lower branch, which was 0·7 inch wide, passed forwards as the shaft of the 2nd rib, and ended in a costal cartilage, which articulated with the side of the sternum at the junction of the manubrium and body. The upper branch was a triangular plate of bone 0·8 inch long, the apex of which was directed

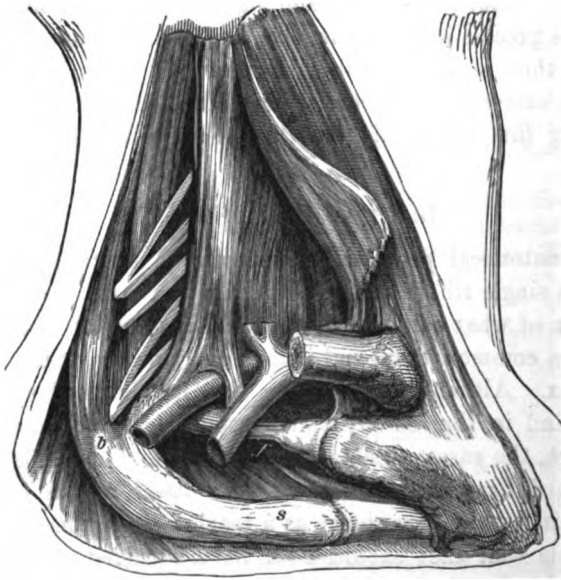


FIG. 1. Right side of neck, displaying relation of soft parts to the unusually formed rib. *b*, The common body; *s*, the anterior part of the shaft of the second rib; *f*, points to the fibrous band of the first rib.

forwards. This plate represented the bony shaft of the 1st thoracic rib, and from it a fibrous band passed forwards, as a continuation of that shaft, to join the attenuated outer end of the 1st costal cartilage, which was articulated to the manubrium immediately below the sternoclavicular joint.

The subclavian artery rested on the triangular bony plate of the 1st rib, which formed the upper bifurcation of the common shaft. The scalenus anticus was split in two near its insertion, and the subclavian

artery passed through the cleft. The part of the muscle in front of the artery was inserted into the fibrous band; the part behind into the upper border of the triangular bony plate. The scalenus medius was inserted into the common shaft; the cords of the brachial plexus emerged between the anticus and medius, and the lowest cord rested in a groove on the upper surface of the common shaft. The scalenus posticus was inserted into the common shaft behind the medius. The subclavian vein rested partly on the tendon of the scalenus anticus in front of the artery, and partly on the fibrous band. The apex of the pleural sac and of the lung ascended into the neck, under cover of the scalenus anticus, for at least one inch above the upper surface of the subclavian artery. The 1st dorsal nerve occupied the short cleft between the necks of the two ribs prior to their fusion. The superior intercostal artery descended in front of their necks as far as the space between the 2nd and 3rd ribs. The vertebral artery ascended to enter the vertebrarterial foramen in the 6th cervical vertebra. The subclavian muscle arose from the cartilage of the 1st rib. The space immediately below this cartilage and the fibrous band was occupied by a plane of muscular fibre, which represented an internal intercostal muscle.

On the *left* side the head of the 1st rib articulated with the side of the body of the 1st dorsal vertebra, and with the disc between it and the 7th cervical. The articulations of its tubercle and of the head and tubercle of the 2nd rib were normal. The shafts of these two ribs fused with each other immediately beyond their tubercles. The greatest breadth of the common body was 1.1 inch; its surfaces were directed obliquely, and a roughness on the outer surface marked the attachment of the serratus magnus. This body did not bifurcate anteriorly, but diminishing in breadth in front of the origin of the fibrous band to 0.8 inch, it ended in front in a costal cartilage, which was jointed with the sternum at the junction of the manubrium and meso-sternum. Springing directly from the inner border of the common body, a little in front of the insertion of the scalenus medius, was a fibrous band, which passed inwards to join the attenuated outer end of the 1st costal cartilage, which had a similar attachment to the sternum as on the right side. Both the right and left blended ribs measured 8 inches from the head of the 2nd rib along the convex border of the common body to the articulation with the costal cartilage.

The scalenus anticus was cleft at its insertion into the fibrous band, and the subclavian artery passed through the cleft. The subclavian vein rested on the anterior division of the tendon. The scalenus medius and posticus and the cords of the brachial plexus corresponded with those on the right side, and the general arrangement of the pleural sac, the superior intercostal artery, and the 1st dorsal nerve was very similar. The interval between the fibrous band and the 2nd rib was the 1st intercostal space, and the plane of muscular fibres which occupied it passed upwards and inwards, and represented the internal intercostal muscle. The subclavius arose from the place of junction of the fibrous band with its costal cartilage. The vertebral artery entered the foramen in the 7th cervical vertebra.

The other ribs were normal. The 12th pair of ribs were each about 4 inches long. The manubrium sterni was transversely elongated, on a level with the first pair of costal cartilages to a breadth of 3·6 inches. Its great breadth was obviously due to an extension of the ossification into these cartilages, so that only a small portion, with which on each side the fibrous band was continuous, remained in the cartilaginous state.

*Case 2.*—Middle-aged man. The vertebral column had the usual formula  $C_7D_{12}L_5S$  Coc. On the *left* side the shaft of the 1st thoracic rib was blended with that of the 2nd rib, so that the common shaft formed by their junction was bicipital. Each of the two heads possessed an elongated neck, a tubercle, and the beginning of a shaft, and the two shafts fused with each other 0·8 inch to the outer side of the tubercle of the rib, and  $1\frac{1}{2}$  inch to the outer side of the body of the 1st dorsal vertebra. The upper head articulated with a shallow fossa on the upper half of the side of the body of the 1st dorsal vertebra immediately above its middle; whilst the lower head articulated with the side of the same vertebra near its lower border, and with the upper

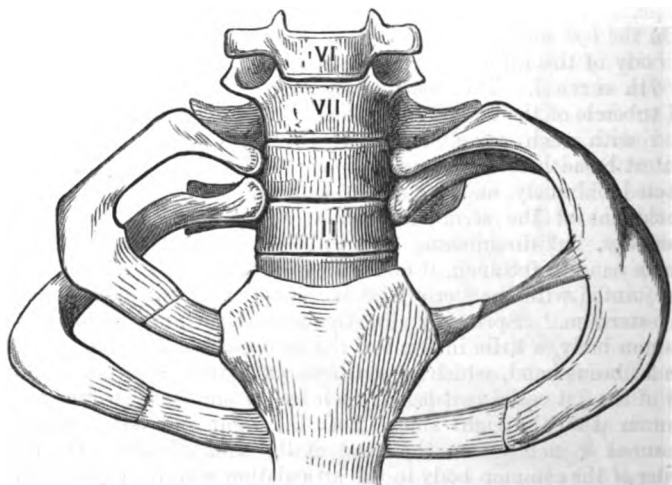


FIG. 2. VI. VII. The sixth and seventh cervical vertebrae; I. II. the first and second dorsal vertebrae. I have not thought it necessary to represent any more vertebrae.

half of the side of the body of the 2nd dorsal vertebra, which are the normal articulations of the 1st and 2nd thoracic ribs. A cleft, 0·2 inch in vertical diameter and  $1\frac{1}{2}$  inch long, separated the 1st and 2nd ribs from each other at their vertebral ends.

The breadth of the shaft of the 1st rib, immediately before the fusion, was  $\frac{1}{2}$  inch, and that of the second was 0·7 inch. After the fusion the common body assumed a breadth at its widest part of 1·3 inch. A faint groove on the upper surface of the conjoined shafts indicated their line of fusion with each other, and on the inner border

2·2 inches anterior to the place of fusion, a rough edge marked where the 1st thoracic rib ceased to take any part in the formation of the common shaft. From this spot a fibrous band arose and arched inwards to join an osseo-cartilaginous bar 1·1 inch in length, which represented the 1st costal cartilage. This structure was attached to the side of the manubrium immediately below the articular surface for the clavicle, and its sternal attachment was 0·8 inch in depth. A plate of bone formed two-thirds of its surface, the rest being cartilage; and its outer end, which was attenuated and continuous with the fibrous band, was quite ossified. The part of the costal arch in front of the place of origin of the fibrous band had a maximum width of 0·9 inch, and belonged entirely to the 2nd rib; it ended anteriorly in a costal cartilage, which was articulated to the side of the sternum at the junction of the manubrium with the body of the sternum.

The fibrous band represented the more anterior part of the 1st rib unossified; and the interval between it and the ossified costal cartilage in which it terminated on the one hand, and the shaft of the 2nd rib and its cartilage on the other, was the representative of the 1st intercostal space. This space was occupied by membrane. The direction of the surfaces of the conjoined shafts was oblique, and the outer surface had a roughness for the serratus magnus. The length of the costal arch, measured from the head of the 1st rib along the convexity of the curve to the place of origin of the fibrous band, was  $4\frac{1}{2}$  inches, and from the head of the 2nd rib to the junction with the costal cartilage the curve was  $8\frac{1}{2}$  inches.

The brachial nerves rested in a groove on the inner border and upper surface of the shaft of the conjoined ribs. The subclavian vessels rested on the fibrous band of the 1st rib. The scalenus anticus was attached to the fibrous band.

The ribs on the *right* side were normal in number and arrangement. The 1st right rib was  $5\frac{3}{4}$  inches long from head along convex outer border to attachment of costal cartilage. The 12th right rib was 3 inches long, the 12th left  $3\frac{1}{2}$  inches. The costal cartilage, both on the right and left sides, had irregular osseous plates developed on their surfaces; this was more especially the case with the 1st right costal cartilage, which had an almost complete bony envelope. No special anatomical peculiarity required to be noted in the cervical or lumbar region.

*Case 3.*—During the time that I was engaged in working out cases 1 and 2, one of my pupils, Mr. Minas S. P. Aganoor of Ispahan, brought to me the right ribs of a skeleton which he had procured at Calcutta some time previously. The first of these ribs possessed two heads, necks, and tubercles, and 0·7 inch external to the tubercles the two shafts were fused together into a common body, and before fusing they were separated by a cleft 0·2 inch in width. The length of the upper division of the rib from head to tubercle was 0·8 inch, that of the lower 0·7. The neck of the lower division was more attenuated than that of the upper. The line of fusion of the two shafts was



marked on the upper surface of the common body by a shallow groove, and the direction of the surfaces was almost horizontal. The greatest width of this body was 0.9 inch. Its upper surface had two grooves for the subclavian artery and vein, and between them was the pointed process for the attachment of the scalenus anticus. The body did not



FIG. 3. Bicipital rib on right side of Case 3

diminish in breadth up to its sternal end; it did not bifurcate, but ended abruptly in the articular surface for the costal cartilage. This rib measured 5.8 inches from the lower of the two heads, along the convex margin of the rib to the sternal end, and the chord of the arc was 2.1 inches.

The rib immediately below the one just described measured 8.7 inches from head to sternal end, along the convexity of the curve, and the chord of the arc was 3.8 inches. It had the characters of a second rib, both as regards the obliquity of its surfaces, its length, and the presence of a rough ridge on the shaft for the origin of the serratus magnus.

The epiphyses of the heads of the ribs were not fully ossified, but those of the tubercles were ankylosed. Unfortunately the other bones of the axial skeleton were not in Mr. Aganoor's possession. From the appearance of the ribs the person was probably neither very muscular nor tall.

Of the nature of the anatomical peculiarity described in cases 1 and 2 there can be no question. That these subjects departed from the normal arrangement in the upper thoracic region was recognised early in the course of the dissection, so that the soft parts were carefully examined as the dissection proceeded, and the ribs, sternum, and vertebræ were preserved. They are undoubtedly, therefore, cases in which the bicipital character of the upper rib is due to a fusion of the shafts of the 1st and 2nd thoracic ribs a short distance in front of their tubercles.

Similar precise data for the determination of the nature of the peculiarity in case 3 were not at my disposal. I had only the

right ribs for examination, and could obtain no information about the sternum, vertebræ, or soft parts. From the characters of the ribs, however, I think it possible to arrive at a satisfactory conclusion respecting the nature of the bicipital rib in this case also.

Case 3 agrees with 1 and 2 in having two heads, necks, and tubercles, and in the shafts being fused into a common body a little beyond the tubercles, so that at first sight it might be thought to have been also formed by a blending of the upper two thoracic ribs. But a closer comparison of the specimens will show that in other respects there are considerable differences between them. In case 3 the direction of the surfaces of the common body is almost horizontal, whilst in 2 and 3 they have considerable obliquity. In case 3 the common body does not bifurcate anteriorly as on the right side of case 1, neither is there any rough edge on its inner border, as in case 2, from which a fibrous band representing the anterior part of the costal arch could have proceeded. In case 3, also, the common body does not diminish in breadth up to its truncated extremity where it joins the costal cartilage, whilst in both 1 and 2 the osseous part of the costal arch undergoes a sensible diminution in breadth prior to the junction.

But what is even more important, the length of the costal arch of the bicipital rib is in case 3 more than 2 inches shorter than in either 1 or 2. To understand the significance of this difference it is necessary to bear in mind that the arch of the normal 1st thoracic rib has a much less span than that of the 2nd. From measurements made in various skeletons, from the head of the rib to the sternal end, I find that the length of the 1st rib, measured along its convex edge, varied between  $4\frac{1}{4}$  and 6 inches, the average being  $5\frac{1}{4}$  inches, whilst the length of the second rib, measured in the same way, varied from  $8\frac{1}{2}$  to  $9\frac{1}{4}$  inches, the average being  $8\frac{3}{4}$  inches. Now, as the costal arch of the bicipital rib in case 3 was only 5.8 inches, whilst in cases 2 and 3 it measured 8 inches and upwards it is clear that in 3 it approximated in its dimensions to the 1st thoracic rib, and in 1 and 2 to the 2nd thoracic rib. Moreover, in case 3 the rib which immediately succeeded the bicipital rib had the length, obliquity of surface, and muscular markings

of a 2nd rib. The conclusion, therefore, to which I have come of the nature of case 3 is this, that the lower head, neck, tubercle, and corresponding part of the common body are the 1st thoracic rib; whilst the upper head, neck, tubercle, and part of the common body belonging to them are a cervical rib, the shaft of which has fused with the shaft of the 1st thoracic rib.

The study of these cases has led me to re-examine the literature of bicipital ribs, and to reconsider the opinions expressed by other anatomists on their nature, and by myself in the paper "On the So-called Two-headed Ribs" already referred to.

The first case to be recorded was communicated in 1740 to the Royal Academy of Sciences in Paris by M. Hunauld.<sup>1</sup> He figured the bodies of two vertebræ with the right ribs attached to them, and stated that he had in his possession—

"The skeleton of an adult in which the 1st rib on each side, well formed posteriorly and articulated with the 1st dorsal vertebra, joins and fuses with the 2nd rib, which by this union becomes larger than usual."

Sandifort has also figured<sup>2</sup> from the Museum at Leyden the first right rib of an adult man "quæ duo plane distincta capita habet." Like Hunauld's specimen it closely corresponded in the place of fusion with my cases; it diminished in breadth towards its sternal end, and the spot where the fibrous band had probably been attached was marked by a short process.

Dr. Robert Knox, in an important memoir on the cervical ribs in man,<sup>3</sup> reproduced M. Hunauld's figure and description, and described and figured some specimens in his own collection. In commenting on Hunauld's specimen Knox expressed, though with some hesitation, the belief that it might not be an example of fusion of the 1st and 2nd thoracic ribs, but of a cervical rib blended with the 1st thoracic, and he also gave a similar interpretation to Sandifort's specimen. Halbertsma, in a subsequent memoir,<sup>4</sup> has expressed the same opinion of the Leyden

<sup>1</sup> *Op. cit.*, p. 535, fig. 2.

<sup>2</sup> *Museum Anatomicum*, tab. xlix. figs. 1 and 2.

<sup>3</sup> *London Medical Gazette*, November 3 and 10, 1843.

<sup>4</sup> *Archiv f. d. Höllandisch Beiträge z. Natur. u. Heilk.*, Bd. 1, 1858, quoted by Gruber.

specimen. Knox figured a specimen in his own collection (figs. 2 and 3) as a cervical rib blended with the 1st thoracic. In fig. 5 he represented another specimen which may, he thinks, be analogous, though, as he had no knowledge of their sternal and vertebral attachments, they "may be a partial fusion of the 1st and 2nd thoracic ribs." It should be stated, indeed, that neither in Hunauld's, Sandifort's, nor Knox's specimens is any information given of the sternal attachment of the ribs, and it is in Hunauld's case only that the vertebral attachment is stated. As Knox's specimens are now in the Anatomical Museum of the University of Edinburgh, I am able to refer to them and give some further account of their characters. The one drawn in his figs. 2 and 3 is very like Sandifort's specimen. The common body is 1·2 inches broad, and a short process springs from its inner border, to which had probably been attached a fibrous band similar to that described in my cases 1 and 2. In front of this process the shaft of the rib diminished to 0·8 inch in breadth, and had a truncated end for the costal cartilage. The surfaces of the common body were oblique; on the outer surface was a roughness, probably for the serratus magnus, and the part which belonged to the upper of the two elements of the rib was grooved for the subclavian artery. The length of the lower element was  $7\frac{1}{2}$  inches, that of the upper, as far as the process above referred to, was  $4\frac{1}{4}$  inches. The common body of the ribs drawn in his fig. 5 had the unusual breadth of 1·8 inches, and it retained this breadth up to its anterior end, where it bifurcated. The upper branch joined almost immediately the costal cartilage, the lower was one inch long and seemed as if a piece had been broken off its end. The surfaces were oblique, and a rough mark on the outer surface was probably for the serratus magnus. The upper part of the common body was grooved for both the subclavian artery and vein, and between these grooves was the scalene tubercle. The upper element of this bicipital rib was  $5\frac{1}{2}$  inches long, the lower  $7\frac{1}{2}$ , though it had probably been longer when complete.

Huntmüller described some years ago<sup>1</sup> a preparation in the Blumenbach collection in Göttingen, which he states to be like Sandifort's specimen, an example of a cervical rib fused with the

<sup>1</sup> *Hentle u. Pfeufer's Zeitschrift*, Bd. xxix. p. 150, 1867.

1st thoracic. The common body is broad in its mid part, but in front of the spot where the part which he regards as a cervical rib terminates, the rib diminishes in breadth. The surfaces are not horizontal, and he states that in its aspect it has lost the characters of the 1st thoracic and presents a transition to that of the 2nd rib. The length along the convexity is 18 cent. (7·087 inches), which, as I have already pointed out (p. 393), is nearer that of the 2nd than of the 1st thoracic rib. Professor Wenzel Gruber, in his elaborate memoir "On Cervical Ribs,"<sup>1</sup> cites Sandifort's and Huntemüller's specimens as examples of cervical ribs fused with the bony shaft of the 1st thoracic.

In my paper on the "So-called Two-headed Ribs" I described and figured from the human subject two specimens in the Ana-



FIG. 4. (Specimen 383a.) *a*, The neck of the upper; *b*, that of the lower of the rib.

tomical Museum of the University of Edinburgh, 383a and 80e, and for facility of reference I reproduce the figures here.

Although I recognised that the upper of the two ribs closely resembled in form the 1st thoracic, and the lower was like a 2nd thoracic, yet by attaching more value to the characters of the two vertebræ preserved in 80e than to the forms of the ribs, I came to a similar conclusion with regard to them that Knox, Halbertsma, and Huntemüller had done with their specimens, and pronounced the upper element to be a largely developed

<sup>1</sup> *Mém. de l'Acad. Imp. des Sciences de St. Petersbourg*, xiii., 1869.

cervical rib, blended with the 1st thoracic. The additional knowledge that I have since gained by the dissection of cases 1 and 2 has led me to reconsider this conclusion. All these four specimens closely resembled each other in the form of the heads and necks, in the place of fusion of the shafts just beyond the tubercles, in the obliquity of the surfaces of the common body, and in the presence of a roughness for the attachment of the serratus magnus. In 80e the upper element measured from head to sternal end  $5\frac{1}{4}$  inches, the lower element  $7\frac{3}{4}$  inches; in 383a the upper element measured  $5\frac{1}{4}$  and the lower 8 inches, so that in these dimensions they approximated to the corresponding curvatures in cases 1 and 2 and to the measurements of normal 1st and 2nd thoracic ribs. In case 2, also, the head

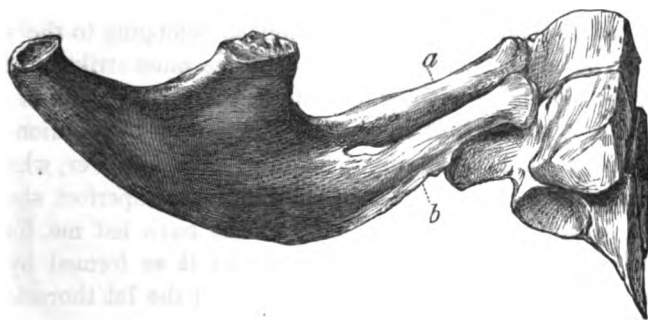


FIG. 5. (Specimen 80e.) *a*, The neck of the upper; *b*, that of the lower of the two ribs. The specimen is represented from below, and the drawing much foreshortened.

of the upper rib articulated as in 80e with the side of the body of its vertebra, near the middle. 80e and 383a differed, however, from 1 and 2 in that the common body, divided anteriorly into two shafts, each ending in a costal cartilage; but, as is shown by the difference on the two sides of the body in case 1, variations in the mode of termination anteriorly of these blended ribs may take place even in the same person, so that this is not to be regarded as an important character.

The nature of the anatomical peculiarity in 1 and 2 is not open to question, for the ribs were all seen in their places, and their connections with the vertebræ and sternum examined. It

is therefore much more probable that 80e and 383a should, from their similarity in appearance, be also regarded as due to a fusion of the 1st and 2nd thoracic ribs, and not of a cervical rib with the 1st thoracic, as I had formerly described. A similar interpretation should also, I think, be given to Sandifort's, Knox's, Halbertsma's, and Huntemüller's cases; and we must accept M. Hunauld's own statement of the nature of his specimen as correct, and discard the view of its being a cervical rib blended with the 1st thoracic, which was suggested by Dr. Knox.

But whilst considering all these cases to be examples of union of the elements of the 1st and 2nd thoracic ribs, I do not imply that a cervical rib, when it occurs, may not sometimes have its bony shaft fused with the 1st thoracic rib, although undoubtedly such cases are extremely rare. If I am right in regarding the above cited cases, which have by so many observers been described as examples of such fusion, as belonging to the same category as my specimens 1 and 2, then we must strike out from anatomical literature those examples which it has been customary to quote as illustrating the junction by bony union of a cervical with a thoracic rib. The third case, however, which I have described (p. 391), notwithstanding the imperfect state of the skeleton, presents such characters as have led me, for the reasons already given (p. 393), to regard it as formed by the fusion of the shaft of a cervical with that of the 1st thoracic rib.

#### BICIPITAL RIBS IN WHALES.

In my previous paper on this subject I referred to the descriptions which have been given by Dr. J. E. Gray, Professor van Beneden, and Professor Flower of skeletons of the Cetacea, in which the bicipital form of the first rib had been seen, and I at the same time described a specimen which I had obtained in a miscellaneous collection of whales' bones from the Cape of Good Hope.

Since then I have met with another rib of the same kind which illustrates a similar peculiarity. It formed a part of the skeleton of a *Balænoptera*, some of the bones of which were found in 1859, others in 1863, embedded in clay in Christie's brickfield, in the carse land near the town of Stirling, and about 100 yards from the bed of the river Forth. They were lying in the "blue

slink," from 13 to 14 feet below the present surface, and from 3 to 4 feet above the present high-water mark.

The rib was cleft at its vertebral end by a fissure  $5\frac{1}{2}$  inches deep, at the bottom of which the two segments fused together into a common shaft, flattened on its two surfaces. Each segment was a flat plate of bone truncated at its vertebral end, and the one was both in width and length about  $\frac{3}{4}$  inch greater than the other. The line of fusion of the segments was marked on one surface, and on the inner border of the common body, by a shallow groove. Each segment had an elongated, tubercle-like roughness on its upper border, immediately before the rib made its downward curve, which on the longer segment was before, but on the shorter after, the fusion had taken place. The common shaft was from 4 to  $4\frac{1}{4}$  inches broad up to its sternal end, where it widened out to nearly 7 inches. The length of the rib from the vertebral end of the longer segment along the convexity of the curve to the sternal end was 3 feet 7 inches. The skeleton was so imperfect, and so many of the bones were broken, that it was not possible to ascertain with which vertebræ this rib had articulated.



FIG. 6. Two-headed rib of *Balænoptera* embedded in the carse clay. This figure, and figs. 1, 2, and 3, have been drawn on wood by my son, Mr W. A. Turner.

As it is the 1st rib which in the Cetacea has been seen to have this bicipital or bifid arrangement at the vertebral end, it has been customary to regard it as due to the fusion of a supplementary cervical rib with the 1st thoracic. That cervical ribs do occasionally occur in the Cetacea, as in man, has been shown, especially by Professor van Beneden, who has described examples in skeletons of *Balænoptera laticeps*, *Delphinus delphis*, and *Phocæna communis*.<sup>1</sup> In some of these the cervical rib was

<sup>1</sup> *Bulletin de l'Acad. Roy. des Sciences de Belgique*, xxvi. p. 7, 1868.



not fused with the thoracic ; but in one very remarkable specimen from the skeleton of a *Balænoptera laticeps* from the North Cape, whilst on the right side the cervical rib was free so that the 1st thoracic rib was not bifid, on the left side a longitudinal bar of bone formed an eminence on the anterior surface of the 1st thoracic rib, which Van Beneden interprets as a cervical rib fused with the 1st thoracic.

From the fact which has now, by the dissection of my cases 1 and 2, been satisfactorily proved, that in man a similar bicipital appearance may arise from a fusion of the 1st and 2nd thoracic ribs, the question naturally emerges, May not a like fusion also take place in the Cetacea ? so that it is possible that some of the specimens of bifid first ribs, so common in skeletons of *Balænoptera laticeps*, may be due to this cause, and not to a fusion of a cervical with the 1st thoracic rib. In connection with this view I may refer to my description of the skeleton of *Balænoptera borealis* (*laticeps*), published last year in this *Journal* (vol. xvi., April 1882, p. 479), in which no supplementary cervical rib was present, but where the 1st and 2nd right thoracic ribs were fused together at their sternal ends.

Although the general conclusions arrived at in my former paper on bicipital ribs are in the main unaffected by the additional facts now recorded, they require modification in one or two particulars, and may be re-stated briefly as follows :—

1st. In both Man and the Cetacea cervical ribs are occasionally developed in connection with the 7th vertebra.

2nd. In both the cervical ribs may remain free or be fused with the 1st thoracic rib, so as to make it bicipital.

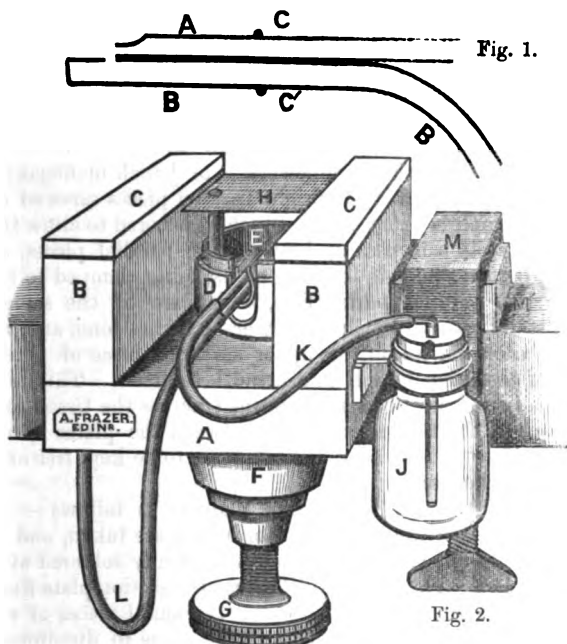
3rd. In Man a similar bicipital form may be due to fusion of the shafts of the 1st and 2nd thoracic ribs with each other at their vertebral ends, and it is probable that this may also occur in the Cetacea.

4th. In either of the forms of fusion specified in 2 and 3, the two limbs, into which the vertebral end is divided, lie in different transverse planes, and the bifurcation is due to the partial fusion of two morphologically distinct rib-elements.

5th. The presence of a cervical rib, or the bicipital form of the 1st rib, is only an individual peculiarity, and is not to be regarded as affording any evidence of either specific or generic difference.

**NEW FORM OF ETHER MICROTOME.** By CHARLES W. CATHCART, M.B., F.R.C.S., *Lecturer on Anatomy, Surgeons' Hall, Edinburgh.*

IN venturing to add another to the many forms of Freezing Microtome which have been produced of late, I may begin by explaining what objects I had in view in devising the present apparatus, as a preliminary to describing its mechanism. These objects, briefly, were—(1) to obtain a simple ether spray producer which would not allow any ether to escape unevaporated; (2) to have an efficient microtome for use with the ether spray, which would be so simple in its mechanism as to admit of manufacture and production at a comparatively low cost.



After a considerable amount of time and trouble, I have succeeded in producing a Microtome which can be sold at 15s., including the spray producer, and which freezes  $\frac{1}{4}$  of an inch of tissue in  $1\frac{1}{2}$  or 2 minutes, using in the process about 2 drachms of ether, which cost something less than a farthing. The instrument may be described under the heads of (1) the Spray, (2) the Microtome.

The spray producer (fig. 1) works on the same principle as the scent sprays which have been in use for a long time, where a jet of air playing across the top of a tube draws up the fluid from its interior by tending to make a vacuum in it. The bellows used are the ordinary hand ones

sold for carbolic and other spray producers, these being as cheap and efficient as any that can be got. In working at the spray points, I began by selecting the size of air-hole that these bellows could easily feed with a continuous blast of air, and then, experimenting with various sizes of vaccine tubes, I found at last, that with the smallest size I could produce a spray which, at about half an inch's distance from any object, contained just as much ether as the given blast of air could evaporate. There was then of course no running of the ether to waste, while at the same time an intense cold was very rapidly produced. The method of adapting the spray points to one another is a modification of the ordinary one, and is adopted from a German model. It is as follows:—Two fine brass tubes are taken—one (A) is brought to the requisitely fine point for the ether, and the other (B), being closed at the end, has the air-hole bored at the side, a little behind the closure. The point of the ether tube is then placed over the middle of the air-hole, and the tubes, laid one over the other, are soldered together in this position; the free ends of the tubes are then connected with the air bellows and the ether bottle respectively by means of india-rubber tubing, and this part of the apparatus is complete.

The microtome (fig. 2) consists of the framework, and the mechanism for raising the section. The framework is of  $\frac{1}{2}$ -inch mahogany, and is in the form of a base with two upright parallel pieces screwed on to it. The base (A), which is about  $2\frac{1}{2}$  by 4 inches, is bored to allow the tubes for raising the section to pass up between the parallel pieces, and has a projecting part at one side to allow of its being clamped to the table (M'). The two parallel parts (BB), which are of the same  $\frac{1}{2}$ -inch mahogany, stand about  $1\frac{1}{4}$  apart; they are 4 inches long, and, rising to 1 inch high, each carries on the upper surface a piece of  $\frac{1}{4}$ -inch plate glass (CC) of the same length and breadth as itself. This is to support and steady the knife as it is pushed across the tissue to be cut, while the fact of the tissue coming up between the plates allows that part of the knife which is to cut the specimen to be kept free of contact until it touches the tissue.

The method of raising the section plate is as follows:—About 2 inches of accurately-fitting double brass tubing are taken, and into the outer one (D) the nut (F) of a fine screw is firmly soldered at what is to be its lower end. The inner tube (E) has the section plate fixed to its upper end by two screws, with, however, two small pieces of vulcanite intervening between the plate and the tube, so as to disconnect them as much as possible, and into the lower end of the inner tube a transverse bar is fitted, against which the screw coming through the outer tube presses when it is desired to raise the section plate to which the inner tube is attached. By means of a small screw-nail fixing the outer screw to the bar in question, the inner tube can be withdrawn, as well as pushed up whenever that movement is required. A milled head (G) has been substituted for the ordinary capstan arms, for turning the main screw round.

The spray points are introduced at the requisite distance below the section plate by cutting a narrow slot through both tubes, and fixing to the inner one a piece of bent brass, into which the spray points can be

pushed and held firmly, while a small shoulder on the latter prevents them from passing beyond the centre of the under surface of the plate.

Finally, the ether bottle (J) is fastened to the side of one of the up-right pieces of the framework by a simple hook and eye, the hook being fixed to a collar round the neck of the ether bottle, and the eye to the side of the framework in question. It will be seen, I think, from this description, that with the exception of the fine screw for raising the tissue, the details of the mechanism are very simple, hence the low price at which it can be sold; and in practice it has been found to work admirably.

The instrument can be had from Mr. Fraser, optician, 7 Lothian Street, Edinburgh, who has most carefully carried out my suggestions as we went along, and who has suggested a part of the mechanism himself.

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#### A CASE OF RIGHT-SIDED SIGMOID FLEXURE AND RECTUM. By ERNEST E. MADDOX, M.B., *Res. Phys.*, *Royal Infirmary, Edinburgh.*

THE lower part of the large intestine was observed to take the following abnormal course during the *post-mortem* examination of a male subject in the Royal Asylum, Morningside.

For permission to communicate it, I am indebted to the courtesy of Dr. Clouston.

For some inches below the splenic flexure the descending colon occupied its usual position, but thence, with rather an abrupt curve, it passed across the abdomen to the right side, and lay, in this horizontal part of its course, behind the small intestines and the origin of the mesentery, bound by peritoneum to the aorta and vena cava near the termination of the former.

It was then continued into an ample sigmoid flexure in the right iliac fossa, which lay in front of the cæcum, and was invested by peritoneum derived from the covering of that viscus through a meso-colon about two inches wide.

The rectum coursed down the right side of the sacrum, and but for this reversal of position bore its normal relation to the peritoneum.

This abnormality must from its nature have been congenital, and differs essentially from the not infrequent displacements of the sigmoid flexure to the right, attributed to the exceptional length of the meso-colon, and in which the descending colon must of necessity lie *in front* of the small intestines, to reach the right iliac fossa. Their occurrence must be viewed as more or less accidental, and due to the extreme licence afforded to the sigmoid flexure by the unusual length of its tether.

The present case would be of much interest clinically, for the diagnosis of a large tumour in the right iliac fossa, with no swelling in the left, would remove any suspicion of impacted fæces in the sigmoid flexure, and the presence of another tumour in the middle line, bound closely to the aorta, and perhaps transmitting its pulsations, would render a correct interpretation of the physical signs still more improbable.

NOTE to Mr. HASWELL's Paper on "*Some Points in the Myology of the Common Pigeon.*"

[After the issue of our January number containing Mr. Haswell's paper, we received from him, from Sydney, the following note, which he requested us to substitute for the last paragraph, see p. 221, but as it arrived too late for that issue, we now insert it. In his accompanying letter he states that he has now had the opportunity of dissecting anemu, and that it possesses the so-called lumbricalis muscle.—EDITOR.]

*The Presence of Lumbricales in the Foot.*—Dr. Gadow states that the muscle which I have ventured to regard as the homologue of the lumbricales of mammals is found in many other birds, "e.g., the Ratitæ," and has been described by Meckel in his *Vergleichende Anatomie* and his *Archiv. für Anatomie u. Physiologie*. Not having been able to consult the volumes referred to here, I am unable at present to check Dr. Gadow's identification of the muscle in question with one described by Meckel. I have no doubt, however, that what he says on this head is correct. The statement, on the other hand, that the muscle occurs in many other birds, is certainly erroneous. It has been entirely overlooked by more recent writers; no mention is made of it by Owen in his *Memoir on the Apteryx*, in his article "Aves" in Todd's *Cyclopædia*, or in his *Comparative Anatomy and Physiology of Vertebrates*, nor by Selenka in the *Vögel* of Bronn's *Thierreich*, nor by Alix in his *Appareil locomoteur des Oiseaux*, nor is any allusion made to it in any of Garrod's papers, and this is particularly significant in the case of the only Ratite bird—the ostrich, of which he gives a detailed account of the flexors of the toes. I may add that I have examined representatives of many families of birds for this muscle, and have failed to find it in any save the pigeons, the fowls, and the emu. Thus, though not peculiar to the pigeons, it is a highly characteristic muscle, and though it may have been observed by Meckel, I can claim at least the merit of its re-discovery after a long period of neglect at the hands of those who have more recently written on avian myology.

W. A. HASWELL.

*The concluding part of Dr. Huy's memoir on "The Action of Saline Cathartics," will appear in the July number.*

ERRATA in Dr. DOBSON's Paper on the "*Homologies of the Long Flexor Muscles.*"

Vol. XVII.—January 1883.

- P. 145, line 8 from top, third word from left, for "superficial," read "deep."  
 P. 168, line 18 from top, second word from left, for "these," read "the."  
 P. 168, line 24 from top, second word from left, for "dorsatus," read "dorsalis."  
 P. 179, line 28 from top, second word from left, for "fibularis," read "tibialis."

# Journal of Anatomy and Physiology.

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THE ACTION OF SALINE CATHARTICS. By MATTHEW HAY, M.D., *Demonstrator of Practical Materia Medica in the University of Edinburgh.*

(Continued from page 248.)

## CONCLUSION.

It is now desirable that I place together in a readily intelligible form the more important results derived from the numerous experiments which I have made for the purpose of ascertaining more precisely the nature of the action of a saline cathartic. This is the more necessary, as, so far, the results of each series of experiments have been mostly considered by themselves, and not in conjunction with those of the other series.

In my first experiments, it will be remembered that I hoped to prove, and succeeded in proving by the method employed, that the salt does not cause any increase of secretion within the alimentary canal, but hinders the absorption of the fluids in which it is dissolved, being itself slowly diffusible and uniting with and retaining the water. That theory I was compelled to abandon after obtaining the results of the three subsequent series of experiments. The work upon which it was based is not, however, valueless, for, besides being accompanied by certain interesting analyses exhibiting the distribution of the salt in the urinary and fæcal excretions, it has shown that the secretion which the purgative excites is greatly diminished if the blood is previously concentrated; and, indeed, is so much diminished that in most cases purgation does not at all occur. It therefore rests with me to construct another theory which will meet the requirements of the results of all of the experi-

ments and unite them into a harmonious whole. I shall, for the sake of simplicity and lucidity, consider these results in relation to the following points and in the following order, supplementing them, where necessary, by other experiments.

**A. The Effect of the Saline Cathartic on the Alimentary System.**

I. Does the salt provoke an increase of secretion within the alimentary canal?

II. The source of the secretion :—

1. Stomach.
2. Liver.
3. Pancreas.
4. Small intestine.
5. Large intestine.

III. The nature and characters of the secretion.

IV. The mode of the production or excitation of the secretion.

V. The action of the salt on the peristaltic movements of the intestines.

**B. The Effect of the Saline Cathartic on Other Parts of the Body than the Alimentary System.**

I. On the blood and circulation.

II. On the urinary secretion.

**A. THE EFFECT OF THE SALINE CATHARTIC ON THE ALIMEN-  
TARY SYSTEM.**

*I. Does the Salt provoke an increase of Secretion within the Alimentary Canal?*

The experiments of Series B., C., and D. answer this most distinctly in the affirmative. Some objections might be urged against the method employed in Series B., but the methods of Series C. and D. are perfectly unimpeachable; and I claim by these to have completely and satisfactorily demonstrated for the first time that the salt does actually excite secretion. In the experiments of Series B., by using a certain proportion of a 10 per cent. solution of sulphate of soda to a given length of intestine, it was found that a solution of this strength did not increase in bulk. The quantity of solution was, however, probably smaller than what at any moment comes in contact with

the same length of intestine during the action of a purgative dose of the salt, administered in the usual manner; for, in Experiments LV. and LVII. (Series C.) and LIX. (Series D.), a 5 per cent. solution appears to be that which neither increases nor diminishes in bulk. But, apart from the increase of the fluids in the alimentary canal, I am satisfied from the alteration in their characters and properties that even the weakest saline solution provokes secretion, although absorption, proceeding at an equal or greater rate, may prevent any increase in the volume of the fluid within the canal.

## II. *The Source of the Secretion.*

From what part of the alimentary canal is it derived? Is it from one or other, or a combination, of the various portions of the canal and its attached glands; and, if from a combination of some or all of them, to what extent does each contribute?

1. *Stomach.*—If this organ contains no food, the salt does not remain long within it, probably not over half an hour (Experiments LXII., LXIII., and LXIV.). During this short period a 10 per cent. solution will scarcely increase in bulk (Experiments LXIX. and LXX.), while a 20 per cent. solution may excite a fairly rapid secretion (Experiment LXII.). As the salt is not usually administered in a solution of greater strength than 10 per cent. we may conclude that the purgative extracts but very little fluid from the stomach, and that the secretion excited appears always to possess an alkaline character.

2. *Liver.*—In all the experiments of Series D. the secreted fluid was examined to ascertain if much bile was present, and the fulness or emptiness of the gall-bladder noted. Rutherford<sup>1</sup> has shown that the secretion of the bile is augmented during the action of sulphate of soda, but not during the action of sulphate of magnesia. In most of my experiments with the former salt there was hardly any visible staining of the intestinal fluid with bile, and the gall-bladder was almost invariably well filled; from which I infer that, whatever be the action of the absorbed salt on the secretion of bile by the liver, the discharge of the bile from the gall-bladder into the intestine is little or not at all accelerated by this salt. Even were the discharge increased, it could not add much to the total quantity of fluid within the

<sup>1</sup> Rutherford, *op. cit.*, p. 67.



intestines. For, according to Bidder and Schmidt,<sup>1</sup> the hourly amount of biliary secretion in a cat, 3 kilogrammes in weight, does not exceed 1·8 c.c.; and as in an hour the maximum of the general secretion excited by the purgative has been reached, the bile, allowing for an increase in its discharge, could not contribute more than a very few cubic centimetres of the 40 to 80 c.c. observed to be poured into the intestine during the action of the salt solution (Experiments LX. and LXI.).

3. *Pancreas*.—It is much more difficult to ascertain to what extent this gland adds to the purgative secretion. The pancreatic juice being without colour, its admixture with the purgative fluid cannot be so readily detected as in the case of the bile. Its powerful digestive properties might give a clue to its presence, but as these in their nature are identical with some of the digestive properties of intestinal juice, its presence cannot with certainty be proved, especially if in small quantity. If in large quantity, the difficulty is not so great, for its diastatic and proteolytic powers are very much stronger than those of the intestinal juice. I have compared the diastatic power of the intestinal secretion obtained by the action of the salt within a washed out ligatured loop—and which was, therefore, uncontaminated with pancreatic juice—with the fluid found in the intestines after the action of the salt administered per os. Five cubic centimetres of the former (Experiment XLIX.) produced 0·489 gramme of sugar from a 1 per cent. solution of starch, while the same quantity of the latter (Experiment LXI.) produced 0·662 gramme. Moreover, 5 c.c. of the blood of the cat used in the latter experiment contained sufficient ferment to form 0·411 gramme of sugar from a starch solution. We therefore infer that a saline purgative scarcely, if at all, stimulates the outflow of the pancreatic juice. For, had the fluid of the intestine in Experiment LXI. contained much of this juice, the diastatic power of the fluid would have been very much greater than that represented by the quantity of sugar obtained from the starch digested. The digestion in each case was continued for forty-eight hours. Kroeger<sup>2</sup> found that 1 c.c. of pure pancreatic juice could in the course of half an hour convert 4·67 grammes of starch into

<sup>1</sup> Bidder u. Schmidt, *Die Verdauungssäfte*, &c., Leipzig, 1852, S. 209.

<sup>2</sup> Kroeger, *Hermann's Hdbuch. d. Physiologie*, Bd. v. Th. 1, Maly, S. 195.

sugar, and Roberts, in his *Lectures on the Digestive Ferments*, assigns to the juice an almost incredibly high diastatic power<sup>1</sup>; and there is no reason to believe that the secretion in the cat is not as active as that obtained from other animals. We accordingly conclude that the pancreatic juice shares but very slightly in the increase of the purgative fluid within the alimentary canal, as its presence even in very small quantity would impart a high diastatic power to the purgative secretion. It is but right to mention that Barbier<sup>2</sup> quotes Gendrin as stating that he had experimentally determined that the pancreatic secretion is excited during the action of a purgative. The name of the purgative is not however given.

I might make use of the same form of argument as I have done in the case of the bile to prove that, even were the pancreatic secretion stimulated, the quantity poured out in the course of an hour would be perfectly insignificant compared with the observed increase of the purgative fluid within the intestines during that time. The pancreas of the dog during digestion secretes 0.1 c.c. of juice for each kilogramme of the animal's weight<sup>3</sup>; and if the rate of secretion be similar in the cat, about 0.3 c.c. would be poured out in a cat weighing 3 kilogrammes, and, allowing this to be doubled or even trebled by the action of the purgative, it is evident that the pancreas could not add more than 1 or 2 c.c. to the intestinal contents in the course of an hour.

4. *Small Intestines*.—This viscus appears to be the main source of the fluid. Its capability of furnishing a copious secretion under the action of the salt, many of my direct experiments on the intestines (Series B. and Experiment LXXI.) indubitably prove. It is probable that this secretion is rapidly poured out and that it very quickly passes onwards with the salt to the colon, particularly if the intestine be tolerably free from food (Series D., especially Experiment LXIII.). That the characters of the fluid point to its being derived from the intestine, has already received confirmation in the discussion of the almost passive part which the stomach, liver, and pancreas play in its produc-

<sup>1</sup> Roberts, p. 35.

<sup>2</sup> Barbier, *Traité de matière médicale*, 1830, tom. iii. p. 194.

<sup>3</sup> Bidder u. Schmidt, *op. cit.*

tion, and will be further supported when we pass to the actual consideration of these characters.

5. *Large Intestine*.—It is somewhat difficult to determine with accuracy how much of the fluid is contributed by the large intestine in a case of ordinary purgation. For, by the time that the salt has reached the colon, it has become so diluted by the secretion of the small intestine as no longer to possess its previously active excito-secretory power. But that the large intestine is capable of furnishing a fair amount of secretion, if sufficiently stimulated by a tolerably concentrated solution of the salt, is supported by several of the experiments of Series B. It is possible, indeed highly probable, that, even when the salt reaches the colon in a diluted condition, and when the fluid undergoes no increase within that viscus, there is effected, by an equality of absorption and secretion, a considerable admixture of colic secretion with the purgative fluid.

The general conclusion, therefore, as to the source of the purgative fluid, is that it is mainly derived from the intestines, and particularly from the small intestine; the stomach, liver, and pancreas supplying under ordinary conditions very little or practically none of the fluid.

### III. *The Nature and Characters of the Secretion.*

If it is granted that the secretion excited by the presence of the salt is derived almost entirely from the mucous membrane of the small intestine, it still remains to be decided what is the nature of the secretion. Among those who admit that the purgative does excite secretion there is much difference of opinion as to this, some maintaining that it is a true succus entericus, others that it is an exudation, and others that it is a transudation. Before proceeding to determine to which of these forms of secretion the purgative fluid is by its characters most closely allied, it will be well to briefly define what we understand by each of these varieties of secretion.

An exudation is a fluid which is poured out by the vessels of a tissue in a state of inflammation produced by irritation or otherwise. Of all the fluids met with in the body and outside the blood and lymph vessels, it is that which most approaches in composition the blood-serum; and, like the serum, it contains a large amount of organic matter, particularly of coagulable

albumen, and it abounds also in leucocytes. Further, the inflamed condition of the tissue producing the exudation is generally evidenced by the unusual distension of its blood-vessels, and consequent redness.

The term transudation is often indifferently applied to an inflammatory or a dropsical effusion. Here, however, we shall use it in its stricter sense, and apply it only to a fluid possessing a dropsical character, which may be, and most commonly is, secreted independent of any inflammation of the organ or tissue from which it proceeds, and is generally the result of changes in the pressure or composition of the blood.

The transudation resembles the exudation qualitatively in its composition, but contains much less organic matter. Both fluids yield a very distinct coagulum of albumen when heated, especially the exudation. Apart from the circumstances which occasion their production, and which are often difficult to determine, the exudation and the transudation are mainly to be distinguished from each other by the difference in the quantity of organic matter they contain. And it is relying upon this difference, and on the presence or absence of inflammation of the intestinal mucous membrane, that certain previous observers have regarded the purgative secretion as being of the nature of an exudation or of a transudation.

The succus entericus, or intestinal juice, is a secretion whose characters are very imperfectly known. This arises from the great difficulty experienced in obtaining it pure and in sufficient quantity to permit of its analysis. Thiry gives an analysis<sup>1</sup> of some juice obtained by prolonged mechanical stimulation of an intestinal fistula, made by an operation which I have already described;<sup>2</sup> but as the fistula behaved towards purgative salts in a manner contrary to what we now know to be natural, we may conclude that the secretion thus obtained was as much an exudation from irritation as a true intestinal juice, and its chemical composition supports this view. Moreau procured a copious secretion from a loop of the intestines after complete division of its mesenteric nerves, and notwithstanding Vulpian's objections, it is probable from various reasons, some of which I

<sup>1</sup> Hoppe-Seyler's *Physiologische Chemie*, S. 273.

<sup>2</sup> *Supra*, vol. xvi. p. 250.

have already given, that this secretion is a true succus entericus,<sup>1</sup> and not a mere exudation or transudation. This opinion has quite recently received support in a paper on intestinal digestion by Dr. Meade Smith.<sup>2</sup> Dr. Smith, by establishing a permanent fistula in the lower part of the duodenum of a dog, and inserting into it an ordinary Bernard gastric cannula, and by occluding the intestine a few inches above and below the fistula by means of two distended india-rubber balls, has, after washing out the portion of gut between the balls, obtained as much as 20 to 40 c.c. of secretion from 12 inches of intestine, and that without the application of stimulation. The secretion thus obtained possesses almost exactly the same composition as that of Moreau. The most remarkable feature of intestinal secretion is that it contains a very small quantity of organic matter—in Moreau's secretion, 0·39 per cent., and in Smith's secretion, 0·54 per cent. We may, therefore, with tolerable safety conclude, in spite of the observations of Thiry and others, that a very small percentage of organic matter is a constant character of intestinal juice. It is upon this distinction that I shall mainly rely for the determination of the nature of the purgative secretion.

Certain other characters have been ascribed to intestinal juice, which ought to help us to distinguish it from an exudation or a transudation. It is said to be able to digest starch, invert sugar, emulsify fat and peptonise albumen. Much variety of opinion, however, exists as to its having any or all of these properties. I shall immediately consider to what extent these properties are possessed by the purgative fluid.

With the object of facilitating the comparison of the purgative

	Exudation. <sup>3</sup>	Peritoneal Transudation. <sup>4</sup>	Succus Entericus.	
			Moreau. <sup>5</sup>	Smith.
Percentage of Solids,	8·15	8·08	1·34	1·14
Organic, . . .	7·25	1·92	0·39	0·54
Inorganic, . . .	0·90	0·79	0·95	0·60

secretion with the exudation, transudation, and intestinal juice, I have thought it desirable to give a precise statement of the

<sup>1</sup> *Supra*, vol. xvi. p. 247 and 248.

<sup>2</sup> Meade Smith, *Medical News*, Philad., April 15, 1882.

<sup>3</sup> Hoppe-Seyler's *Physiologische Chemie*, S. 610.

<sup>4</sup> *Ibid.*, S. 603.

<sup>5</sup> *Ibid.*, S. 272.

percentage of solids in the latter, especially of the organic matter, which consists mainly of albumen.

An acute exudation hardly differs in composition from blood-serum, and varies extremely little with the locality of the body in which it occurs. It is otherwise with the transudation, as the quantity of its organic matter depends largely on the situation of the transudation, being sometimes a little less, but generally much greater, than that contained in a peritoneal transudation, which, in the absence of any analysis of a transudation from a mucous membrane, I have selected partly from its proximity to the intestines, and partly from its representing the class of transudates with a small percentage of organic matter, as offering the only possible standard of comparison for the purgative secretion.

If a comparison be now instituted between these various secretions and the purgative fluid, it will be found that the latter most nearly agrees in composition with the intestinal juice of Moreau or Smith. For, although I very frequently examined it for albumen, I never found more than the merest trace (B. Series of Experiments), as indicated by a *faint* opalescence on acidification and heating, or by the application of other tests for albumen. I regret that it has been impossible to procure exact quantitative analyses of the purgative secretion, as the presence of the purgative salt and of epithelial débris from the intestinal mucous membrane interfered with an accurate determination of the organic and inorganic solids. The quantity of the salt was very large in proportion to the solids of the secretion, and after the evaporation of the fluid it was impossible to be certain that the sulphate of soda, the salt generally employed, and which contains a large quantity of water of crystallisation, was present in its usual form, and not to any extent decomposed, or crystallised with less water. For, from the weight of this residue and of its ash after burning, and from the estimation of the sulphate of soda present, had to be determined the amount of organic matter. Notwithstanding these difficulties, I did on one occasion attempt a quantitative analysis of the fluid obtained from the action of 10 c.c. of a 20 per cent. solution of the salt in a previously well-washed loop of the small intestine, 60 c.m. in length. The cat was killed after three hours, and the fluid

measured 89 c.c. The fluid was filtered to remove from it as well as possible the epithelial *débris*, and was then evaporated to dryness, the residue weighing 3.225 grammes. The weight of the crystalline sulphate of soda recovered was 1.562 grammes, which, after deduction from the total residue, leaves 1.663 grammes, or the amount of solids present in the purgative secretion. Of these solids 0.810 gramme was inorganic, and and 0.853 organic. The percentage, therefore, of organic matter, although higher than that of the *succus entericus*, was not nearly so great as that of an exudation, and was considerably less than that of a transudation. A portion of the organic matter was doubtless composed of the finer epithelial *débris* which might readily have passed through the filter.

As the percentage of inorganic matter in the exudation, transudation and enteric juice is practically the same for all, its percentage in the purgative fluid can afford no help in determining the nature of the fluid. Apart from such an object, it is remarkable, however, that the purgative dejections contained a large proportion of ash, as is apparent from the analyses accompanying the experiments of Series A. and some of Series D. (LIX. and LXVI.); and the larger the interval between the administration of the salt and the occurrence of purgation the greater appears to be the percentage of ash (Experiment XXIII. and p. 281, vol. xvi.).

The large quantity of inorganic matter is probably not due to the secretion, as it is poured out from the intestinal mucous membrane, containing a larger percentage of salts than the blood, but to the condensation, so to speak, of the secreted fluid in the lower part of the intestines by the absorption of its water at a greater rate than of its dissolved salts, in the manner I have already explained in connection with the return of the acid of the purgative salt to the alimentary canal. Whatever be the exact nature of the phenomena involved, we are not warranted in assuming that the poured-out secretion is excessively rich in saline matter, for no secretion contains a larger percentage of salts than the blood from which it is primarily derived. The urine is an exception, but the structural conditions of the secreting organ account for its occupying an anomalous position

Besides the small amount of albumen or organic matter in the purgative secretion, which has been as yet the principal reason advanced for regarding the secretion as consisting of intestinal juice, there is also the presence, frequently in large quantity, of mucus, a substance which one would be more likely to find in a physiological secretion than in an inflammatory exudation. I have more than once in the course of this communication insisted upon this fact as testifying to the true secretory character of the purgative fluid. I do not, however, wish to present this point strongly, as I believe that the mucus forms no essential part of the secretion from the Lieberkühnian follicles, but is produced, in all probability, most largely from the superficial epithelium of the mucous membrane. Moreau's secretion possessed extremely little viscosity.

Another most important fact, and one which strongly weighs against the exudative character of the fluid, is the absence of any trace of inflammation in the intestinal mucous membrane. Many previous observers, particularly Vulpian, have maintained that the salt excites inflammation of the intestinal mucous membrane. But this result they obtained from the injection of a saturated solution of the cathartic into an intestinal loop, an experiment which does not represent what naturally occurs when the purgative is administered per os. Weaker solutions, even a 20 per cent. solution, as my numerous experiments almost without exception prove, never cause the slightest visible redness or inflammation of the intestine.

The microscopical characters of the purgative fluid do not help us much in deciding as to its nature. The fluid usually contained a number of colourless corpuscles, and in some instances they were tolerably abundant, especially in the mucous deposit. If these are to be regarded as leucocytes or lymph-corpuscles, their presence is indicative of the inflammatory character of the fluid. But more probably they are mucous corpuscles; and it may be that they are formed from the protoplasmic contents of the epithelial cells of the villi, which I have generally observed, by microscopical examination, to become goblet-shaped and partially empty after the action of the salt.

I now proceed to consider what evidence there is of the



purgative secretion being a true succus entericus from an examination of its digestive properties. This is a matter of some difficulty for the pharmacologist, as the physiologist has not as yet precisely defined the extent and even the nature of these properties. The more recent investigators assign to it the power of converting starch into sugar, cane-sugar into grape-sugar, and, doubtfully, albumen into peptones, and fat into fatty acids and glycerine; but none of them has attempted to fix the limit of these powers, a point most essential in ascertaining whether or not the purgative secretion consists entirely of intestinal juice or is a mixture of the juice with a large proportion of exudation or transudation. In both cases the fluid would digest starch and sugar, but in the former more powerfully than in the latter. As it is difficult to conceive of even a purely inflammatory exudation from a secreting membrane being unmixed to some extent with the natural secretion of the membrane, a mere qualitative proof of the digestive power of the fluid is of no value; it is necessary also to know the limit of this power. In order, therefore, to provide myself with some data of the absolute strength, as well as of the exact nature of the digestive action of the normal intestinal juice, I made infusions of well-washed intestinal mucous membrane, since it was difficult to obtain the juice itself by ordinary stimulation of the living intestine. Infusions of secreting organs, if not made too dilute, are well known to be often quite as active as the usual secretion of the organ. I also prepared infusions of other tissues and organs of the body, and compared their activity with that of the intestine. Several extensive experiments of this kind were made. The tissue or organ was removed from a fasting animal immediately after being bled to death; it was at once weighed and cut up into minute pieces, and infused for twenty-four hours in four times its weight of a 1 per cent. solution of chloride of sodium. Five c.c. of the infusion were mixed with 10 c.c. of a 1 per cent. solution of starch, and another 5 c.c. with 10 c.c. of a 1 per cent. solution of cane-sugar, all of which were previously ascertained to contain no glucose. The mixed fluids were placed in an oven at a temperature of 35–40° C., and the first appearance of glucose was ascertained by repeated testing with Fehling's solution. To ensure

accuracy many little precautions were necessary which it is needless to detail. Sometimes 5 c.c. of the infusion were mixed with 100 c.c. of a 1 per cent., or with 50 c.c. of a 2 per cent. solution of starch or cane-sugar, and allowed to digest for forty-eight hours, when the amount of digested starch or sugar was quantitatively determined. The animals used were the cat, dog, and rabbit. I shall confine myself for the present to a statement of the results of my experiments with the first of these.

Tissue or Organ infused.	EXPERIMENT CXX.			EXPERIMENT CXXI.	
	Time in which Glucose appeared in		Amount of Sugar obtained from Starch. <sup>4</sup>	Time in which Glucose appeared in	
	Starch Solution. <sup>1</sup>	Cane-Sugar Solution. <sup>2</sup>		Starch Solution. <sup>1</sup>	Cane-Sugar Solution. <sup>2</sup>
	h. m.	h. m.	grms.	h. m.	h. m.
Duodenum, . . .	2 15	never appeared	0·047	1 45	2 30
Jejunum, . . .	2 30	1 45	0·031	2 0	2 30
Ileum, . . .	3 0	12 20	0·019	2 0	4 0
Colon, . . .	2 45	never appeared	0·032	2 30	never appeared
Œsophagus, . .	3 0	„	0·012	...	...
Stomach, . . .	3 0	„	0·018	2 15	never appeared
Pancreas, . . .	at once	„	0·882 <sup>4</sup>	at once	„
Muscle, . . .	3 45	„	0·020	...	...
Blood-serum, <sup>5</sup>	3 0	„	0·035	2 30	never appeared

From these and other experiments I have concluded that the only ferment peculiar to the small intestine is the cane-sugar ferment or the “ferment inversif” of Bernard, and that the intestines do not contain more of the diastatic ferment than is to be found in the tissues and fluids of the body generally. The apparent greater activity which the duodenum exhibits is due to

<sup>1</sup> Five c.c. of infusion mixed with 10 c.c. of 1 per cent. solution of starch.

<sup>2</sup> Five c.c. of infusion mixed with 10 c.c. of 1 per cent. solution of cane-sugar.

<sup>3</sup> Five c.c. of infusion mixed with 50 c.c. of 1 per cent. solution of starch, allowed to digest for forty-eight hours.

<sup>4</sup> Gave no blue colour with iodine.

<sup>5</sup> The serum was, previous to measuring the 5 c.c., diluted with four times its bulk of water.

the presence of a small quantity of pancreatic secretion which has soaked into the mucous membrane, and which the washing has failed to remove. If the intestinal juice contain more diastatic ferment than these experiments indicate, it must be formed in the glands at the moment of its secretion, and cannot be stored up to any extent as are the ferments of the pancreatic, salivary, and gastric glands. But I do not believe that the intestinal glands are exceptional in this respect. The intestinal juice, therefore, is probably not richer than serum in the starch-digesting ferment. The presence, accordingly, of this ferment in the purgative secretion is no proof of the fluid being the intestinal juice and not an exudation or transudation.

Although it would thus appear that an examination of the digestive powers of the purgative secretion, particularly of its diastatic action, will help but little to decide what the nature of the secretion is, yet it will not be altogether uninteresting to consider the results of such an examination. I have tested the digestive powers of a very large number of the purgative secretions. Only those results are of value where the intestine, previous to injecting the salt, has been well washed out, so as to remove every trace of the pancreatic and other secretions. This, however, it is difficult to accomplish thoroughly, as prolonged or vigorous washing is apt to injure the mucous lining of the living intestine. It is to be expected, therefore, that the diastatic action of the purgative secretion will have its power increased by the presence of a trace of pancreatic juice, and that the succus entericus poured out as a consequence of the stimulation of the cathartic, will appear to possess a more powerful diastatic action than the two last experiments might lead us to anticipate. And that this is so the subjoined experiments prove. The proportion of the inversive ferment present is, however, much the same as it was in the infusion of the normal mucous membrane.

The fluids (4) and (5) were both obtained from different loops of the small intestine of the same cat, the former after the action of a 20 per cent. solution of sulphate of soda, the latter after an equally strong solution of chloride of sodium. The mucous membrane of the chloride of sodium loop was much inflamed, and the fluid contained a considerable quantity of albumen. Fluid (6) was obtained by the injection of a 20 per cent.

solution of phosphate of soda, and (7) by the injection of a 20 per cent. solution of sulphate of magnesia. In the first three experiments sulphate of soda was employed, and, in all, the method of the application of the salt was that of Colin and Moreau.

No. of Experiment.	Part of Intestine from which Purgative Secretion was obtained.	Digestion of Starch.		Digestion of Cane-Sugar.	
		Time in which Maltose appeared. <sup>1</sup>	Amount of Maltose formed after 48 hours. <sup>2</sup>	Time in which Invert-Sugar appeared. <sup>3</sup>	Amount of Invert-Sugar formed in 48 hours. <sup>4</sup>
1. (XLVII.)	Ileum	h. m.	grms.	h. m.	grms.
2. (XLIX.)	"	0 30	0·128	6 0	0·081
3. (LII.)	"	0 15	0·489 <sup>5</sup>	...	0·098
4. ...	Jejunum	0 30	...	1 0	...
5. ...	"	0 10	...	2 15	...
6. ...	"	0 15	0·192	2 15	0·076
7. ...	Ileum	0 10	...	0 35	...
		1 30	0·097	2 15	0·189

The secretion provoked within the small intestine by the presence of a saline purgative, contains, therefore, very little of either the amylolytic or the inversive ferment, but probably as much of each as the true intestinal juice would appear to possess, if the digestive powers of a strong infusion of the intestinal mucous membrane may be regarded as fairly representing the digestive activity of the juice itself.

While the comparison I have instituted between the digestive powers of the purgative secretion and those of the intestinal juice offers little or no positive evidence of the former consisting of the latter, yet it presents no objection to the acceptance of this opinion. It is to be remarked that blood-serum contains as much diastatic ferment as the intestinal mucous membrane, and it is probable that an inflammatory exudation will contain as much of the ferment as the serum, from which it is so immediately derived. Accordingly, it must be conceded in support of Vulpian's view, that, were the purgative secretion an exudation

<sup>1</sup> Five c.c. of purgative fluid mixed with 10 c.c. of 1 per cent. solution of starch.

<sup>2</sup> Five c.c. of purgative fluid mixed with 100 c.c. of 1 per cent. solution of starch.

<sup>3</sup> Five c.c. of purgative fluid mixed with 10 c.c. of 1 per cent. solution of cane-sugar.

<sup>4</sup> Five c.c. of purgative fluid mixed with 100 c.c. of 1 per cent. solution of cane-sugar.

<sup>5</sup> Deep blue with iodine at end of forty-eight hours.

instead of true intestinal juice, its diastatic power would be as high as it was actually found to be.

In the protocols of some of the experiments of Series B. and of Series D., there exist some estimations of the fat- and albumen-digesting powers of the purgative fluid, which show that these powers are scarcely, if at all, possessed by the fluid; neither have they been ascribed to the succus entericus, or, if so, only to a very limited extent. Indeed, it is highly probable that the succus entericus is a secretion which, physiologically, is of most service as a solvent and diluent of the food that has already been digested by the action of the gastric, pancreatic, and biliary ferments, and that its digestive function is of the smallest importance, unless for the conversion of cane into grape sugar, the ferment for effecting this change existing nowhere else in the body.

Taking into consideration the various characters, chemical, microscopical, and digestive, of the purgative secretion, which I have given in detail, and remembering that the intestinal mucous membrane presents no trace of inflammation from the action of the salt, the conclusion which I have arrived at is that the secretion is mainly obtained from the follicular glands of the intestines, and is, therefore, for the most part a true intestinal juice. It contains too little albumen, and there is no evidence of inflammation to constitute it an exudation. And it can hardly be regarded as a transudation, even were its characters not other than those which an intestinal transudate might possess; for the conditions which occasion a transudation in the body are always associated with a decided alteration of the composition or pressure of the blood, which alteration cannot be proved to occur after the administration of a saline cathartic, to the extent of causing a rapid localised intestinal transudation.

#### IV. *The Mode of the Production or Excitation of the Secretion.*

This depends considerably on what the nature of the secretion is admitted to be. If it be an inflammatory exudation, as Vulpian seems inclined to hold, then it is produced by the salt irritating and dilating the superficial vessels of the intestinal mucous membrane, causing a stasis of the blood-

current, and an outpouring of serum. This would equally well happen were the salt applied to a mucous membrane devoid of special glands. But I have just stated my reasons for believing that such an inflammatory irritation does not occur.

If, as Schmidt and a few others have suggested, the fluid be a transudation, we must suppose that the salt affects the composition or pressure of the blood so as to allow of a modified serum being transuded through the walls of the blood-vessels of the intestinal mucous membrane. Such an alteration of the blood I have already said does not occur. And, were the transudation the result of the action of the salt on the blood, we would expect purgation to be the most active after the direct injection of the salt into the blood, whereas the experiments of Series E. have distinctly proved that the salt possesses no cathartic action whatever when so injected. But although the purgative secretion cannot therefore be a transudation resulting from the action of the salt on the blood, it may be urged that it is a transudation proceeding from the salt effecting certain peculiar changes in the vessels and epithelial covering of the intestinal mucous membrane. It is very difficult, however, to conceive of such changes, and there is no proof whatever of their occurrence. There is yet another possible method by which the purgative secretion may be a transudation, and which does not necessarily involve any alteration of the structure or function of the intestinal lining. I refer to osmosis, the probability of whose occurrence was first maintained by Liebig and Poiseuille, and afterwards by Funke, Heidenhain, and others. These observers evidently supposed that, with the vessel wall and the intestinal epithelium and the intervening tissue, all acting as a septum, the osmotic power of the salt could come into play and abstract from the blood a fluid or secretion having the characters of a transudate. It is, however, highly improbable that such a septum as this, formed of living tissues, can behave towards saline solutions in the same manner as a septum of parchment or dried bladder. At any rate, I have been able to prove to my satisfaction that the purgative secretion is not the result of the endosmotic action of the salt. What this proof consists of, I shall come to immediately. In the meantime, holding it as

established that the purgative secretion is neither an exudation nor a transudation, but consists of intestinal juice, I shall endeavour to elucidate how the stimulation of the Liber Kühnian glands is effected by the salt.

The salt may excite the activity of the secreting cells of the follicular glands of the intestines, either by coming directly into contact with the cells, or by acting on them reflexly through the agency of the intra-intestinal nerves. Owing to the cells being deeply situated within the gland, and removed from contact with the contents of the intestine, it is not likely that the salt acts directly on the cells. Even were it supposed that the salt could diffuse into the cavity of the gland and thus reach the cells, it could hardly reach the cells more readily than if it had been injected into the blood. For, dissolved in the blood, it would, by means of the blood-vessels of the intestinal mucosa, be brought into the closest proximity to the glandular cells. Yet, as I have frequently had occasion to mention, the salt, although injected into the blood in large quantity, does not excite a drop of intestinal secretion. As little, therefore, may the salt stimulate the secreting cells when it reaches them, and necessarily in a state of dilution, by direct diffusion from the lumen of the intestine. A saline cathartic evidently possesses no special excito-secretory power over the glands of the intestine, such as is well known to be possessed by many other substances, as pilocarpin, the salts of arsenic, antimony, mercury, iron, tin, &c., all of which excite intestinal secretion even when injected into the blood. The saline cathartic, therefore, would appear to stimulate the glands reflexly, by producing a certain impression on the sensory nerves terminating in the surface of the mucous membrane of the intestine, which impression, conveyed, probably through Meissner's plexus, to the secretory cells of the gland, excites them to action; just as certain sapid and other substances can stimulate the salivary glands when brought into contact with the mucous membrane of the mouth.

Besides direct or indirect excitation of the glands, there is yet another way in which it is possible for the salt to abstract secretion from the glands. I once more allude to osmosis. This implies that the salt penetrates the cavity of the gland and establishes osmotic relations with the contents of the secreting

cells, which, as they part with their fluid to the salt, have their fluidity restored by an absorption of liquid from the blood. I have already expressed the opinion that the salt does not penetrate freely into the cavity of the gland, and certainly not in a state of concentration sufficient to exert an appreciable osmotic effect on the secretory cells.

If, then, it be maintained that the secretion excited by the salt is for the most part intestinal juice, and that the outpouring of the juice is the consequence of a reflex stimulation of the glands by the salt applied to the surface of the mucous membrane, it behoves us in the next place to inquire what are the properties possessed by cathartic salts which enable them thus to act, and otherwise to cause purgation.

The group of the saline cathartics consists, as is well known, of certain of the compounds of the alkalies and alkaline earths (usually potash, soda, and magnesia), with mineral and organic acids devoid of intrinsic poisonous action, as sulphuric, phosphoric, tartaric, and citric acids. Indeed, it may be broadly stated that any saline compound, whose acid or base does not possess a strongly specific action, will, if the dose be large enough, produce purgation. But, although the number of possible saline cathartics is thus large, and might include amongst others the alkaline chlorides, nitrates, and acetates, yet the application of the name saline cathartic is restricted in ordinary usage to a few salts, which by experience have been found to purge in a moderate dose and unattended by any disagreeable manifestation of the specific action of the metal or acid of the salt. These salts are the sulphate of soda, sulphate of magnesia, sulphate of potash, phosphate of soda, tartrate and bitartrate of potash, tartrate of soda and potash, citrate of potash, citrate of soda, and citrate of magnesia, the last three being more rarely employed than the others. In virtue of what particular properties, it may be asked, have these salts been found to be more serviceable cathartics than other similar salts without specific action, as, for example, the chloride of sodium and the acetate of potash? It is certainly not on account of any difference in their solubility as compared with other simple salines. Nor can it be entirely due to their being more irritant than other salts towards living tissues, and, therefore, towards the intestinal



mucous membrane. This is an important point, for if the supposition as to the purgative secretion being an inflammatory exudation and the result of irritation were correct, the more irritant salt would be the better purgative. Yet chloride of sodium, although a weaker purgative than sulphate of soda or sulphate of magnesia, is a much stronger irritant. I have more than once injected a solution of chloride of sodium into the loop of a cat's intestine, and observed that it invariably caused considerable reddening and inflammation of the mucous membrane of the loop, and that a large amount of albumen was present in the secretion; whereas a solution of an ordinary purgative salt of the same, or even twice the same, strength caused no redness whatever. The irritant character of the purgative salts cannot, therefore, at any rate wholly account for their peculiar action.

I am strongly inclined to believe that the particular character of a saline cathartic, which enables it so much more powerfully than non-purgative salts to produce such an impression on the intestinal mucosa as to reflexly excite the Lieberkühnian gland, is its *bitterness*. It is remarkable that all the most powerful saline cathartics have a well-marked bitter taste. The most active of all of them is probably sulphate of magnesia, and it is the most bitter. It is a common statement in all text-books on *materia medica* that this salt is not so bitter as sulphate of soda. This is a decided error, for I have made solutions of equal strength of both salts, and have tasted them and asked several of my friends to taste them, and the verdict has invariably been, that the magnesia salt is distinctly more bitter than the soda salt. The sulphate of potash and the neutral alkaline tartrates and double tartrates have all an undoubtedly bitter taste. The phosphate of soda is the only one of the more prominent saline cathartics which forms an exception, and it is well known that it is considerably less powerful than the others. None of the other simple alkaline salts which have not been found to act as efficient purgatives are bitter, as the chlorides, the acetates, and the nitrates. It is generally believed that other secretions of the alimentary canal are strongly excited by bitter substances, and why not also the succus entericus? A bitter substance in the mouth certainly stimulates the salivary flow more strongly than a purely irritant substance; and therapists avail themselves

every day of the commonly accepted opinion that bitters increase the gastric secretion. The bitterness, therefore, of a saline cathartic is probably one important factor in its action. But it cannot be the sole factor, whose possession distinguishes a purgative from a non-purgative salt; for why does such a salt as phosphate of soda, which is practically devoid of bitterness, act at all as a cathartic? It is not that this salt is more irritant than the others. It is probably less irritant. This salt would therefore seem to own some other property which renders it purgative. That property has hitherto been believed to be the high endosmotic power of the salt. And there are many who ascribe the action, in part or entirely, of all saline cathartics to their strong endosmotic action. I have already expressed myself as opposed to this view of the mode of the purgative action of the salt, but as I have not as yet given all my reasons for doing so I shall now state them.

In the first place, were the purgative action of the salt entirely or mainly dependent on its endosmotic power, the salt with the highest endosmotic equivalent would be the best purgative. Aubert and Buchheim, to whose experiments I have already referred,<sup>1</sup> have clearly proved that this is not the case. The endosmotic equivalent of certain salts, or the quantity of water which the salt solution gains for every one part of the salt which it loses when separated from water by a parchment or other septum, was found by Aubert to be as follows:<sup>2</sup>—

	Endosmotic Equivalent of	
	Water-free Salt.	Crystalline Salt.
Phosphate of Soda, . . . .	65	26
Sulphate of Soda, . . . .	25	11
Sulphate of Potash, . . . .	15·9	15·9
Rochelle Salt, . . . .	12·2	9
Sulphate of Magnesia, . . . .	8	4
Acetate of Potash, . . . .	6·5	6·5
Chloride of Sodium, . . . .	2·7	2·7

If these equivalents be multiplied by the purgative dose of each salt the resulting numbers ought to be nearly equal, if the view be correct that the cathartic activity of the salt depends on its

<sup>1</sup> Vol. xvi. p. 245.

<sup>2</sup> Aubert, *op. cit.*

endosmotic power. Aubert has furnished us with such a calculation :—

Phosphate of Soda, . . . . .	1035
Sulphate of Soda, . . . . .	750
Rochelle Salt, . . . . .	297
Sulphate of Potash, . . . . .	277
Acetate of Potash, . . . . .	204
Sulphate of Magnesia, . . . . .	132
Chloride of Sodium, . . . . .	40

I do not quite agree with Aubert in the dose he has assigned to each salt. But even were the dose modified, it would not impair the general conclusion, which cannot fail to be drawn from these experiments, that the endosmotic power of a salt is no measure of its cathartic activity, and that, therefore, endosmosis can at most play only a subsidiary part in promoting the outflow of the purgative secretion. Sulphate of magnesia, the most energetic of all the saline purges, has a very low endosmotic equivalent.

Another reason for distrusting the endosmotic theory is to be found in the B. Series of experiments of this investigation. Attention was there drawn to the fact that, when a 20 per cent. solution of sulphate of soda was injected, much more fluid was secreted, and yet much less salt absorbed, than when a 10 per cent. solution was used. According to the present theory the more fluid there was secreted the more salt there ought to be absorbed. The experiments of Series D. are also strongly opposed to this theory.

My final reason for discrediting the endosmotic theory is based upon an experiment which I made to determine the pressure at which the purgative secretion was poured out into the intestine.

*Experiment CXXII.*—Cat, male, weighing 3·14 kilogrammes, was anaesthetised, and its small intestine was exposed by a mesial abdominal incision in the usual manner. A loop was formed on the intestine by two ligatures about 15 cm. apart, and there was injected into the loop as much of a 20 per cent. solution of sulphate of soda as sufficed to gently distend it. Into the end of the loop was then inserted the end of a tube filled with the same solution and communicating with a mercurial manometer. Care was taken that no fluid escaped at the junction of the tube with the intestine, or at any

other part of the loop or the apparatus. The stimulation of the distension at first caused considerable contractions of the loop, but these after ten minutes cease to occur, and the actual pressure occasioned by the secretion could then be read off. It varied from 21.0 mm. to 23.5 mm. of mercury, and although the experiment was continued for more than an hour, the pressure was never observed to rise beyond the higher of these readings. It may be interesting, for the sake of comparison, to mention that the pressure at which the bile and pancreatic juice are secreted is from 15 to 17 mm.<sup>1</sup>

I next took a piece of the dried intestine of a cat and filled it with the same solution of sulphate of soda, and connected its interior with a mercurial manometer, and dipped it into a  $\frac{3}{4}$  per cent. solution of common salt, as representing the serum of the blood. The pressure of the solution within the intestine rapidly rose to 185 mm., at which pressure the intestine burst.

This experiment, I venture to think, proves very conclusively that the purgative secretion is not the result of the endosmotic action of the salt. For, if it were, how comes it that the pressure of the secretion is so low, and corresponds so closely to the pressure under which other secretions, as the bile and pancreatic juice, are naturally secreted?

If, then, the endosmotic power of a saline cathartic is not a factor in the production of its action, what quality is it of the salt (to return to the question raised) which enables certain of the saline cathartics, although possessed of no bitterness, as phosphate of soda, to act as purgatives? I believe it to be a quality of the salt which, although often existing in the most marked degree in those salts in which the endosmotic power is the strongest, is yet probably in no way related to it. I refer to the greater or less *indiffusibility* of the salt, which is able to help the cathartic effect of the salt, not by aiding the secretion of the purgative fluid, but by hindering the absorption of the fluid after it has been secreted. It is a general statement, accepted even by Aubert and Buchheim, that, other things being equal, the higher the endosmotic power of a salt the greater is its purgative action. But these, and other observers, as Liebig, who maintain that the endosmotic power of the salt can entirely account for its purgative action, have erred in attributing to the endosmotic power what really is due to indiffusibility; and the association of the two qualities in most salts has been to a

<sup>1</sup> Heidenhain, *Hermann's Hdbch. d. Physiologie*, Bd. v.

certain extent the cause of their error. Graham has left us the results of many valuable experiments on the diffusibility of salts, and on looking over these I find that all the purgative salts belong to the less diffusible groups.<sup>1</sup> Phosphate of soda is the most indiffusible; sulphate of magnesia comes next, and at some distance below it stand the alkaline tartrates, which are succeeded by the sulphate of soda and sulphate of potash, and still further down by the alkaline chlorides and nitrates. The order, therefore, of the diffusibility of these salts, although it roughly corresponds to that of their endosmotic power, by no means exactly agrees with it. For the sulphate of magnesia is much more indiffusible than sulphate of soda, and yet is much less highly endosmotic. At the same time, allowing for the varying degrees of bitterness possessed by the salt, this order much more closely corresponds to that of the purgative value of the salts. Indeed, were the bitterness of each salt expressed in degrees, and this multiplied by the number representing the diffusibility of the salt, and the product finally multiplied by the dose, a number would be obtained which, omitting the salts possessing any purely irritant action, would be nearly constant for all the purgative salts.

If it be granted that the greater indiffusibility of a salt is to be associated with an increased purgative effect, it is necessary to inquire in what particular manner this quality of the salt operates. It cannot affect the stimulation of the secretion; it must act solely by hindering the absorption of the secreted fluid. We can readily understand how the salt, being on account of its indiffusibility slowly absorbed by the mucous membrane, presents a mechanical hindrance to the water in which it is dissolved. But does it exert merely a mechanical effect, or in addition, is the indiffusibility of the saline cathartic associated with a stronger attraction between the salt and the water of its solution than exists between other more diffusible and non-purgative salts and water? In the theory of saline purgation advanced as the result of the first experiments of this investigation, but which I have now abandoned, I distinctly assumed the existence of such an attraction or union. The same assump-

<sup>1</sup> Graham, *Chemical and Physical Researches*, edited by Dr. Angus Smith, Edin., 1876.

tion has been made by several previous observers, among whom Buchheim<sup>1</sup> has put it to experimental test by exposing to moist air equal quantities of several salts, purgative and non-purgative, and ascertaining after several days their increase in weight. He found that the less diffusible or purgative salts absorbed on the whole less water than the more diffusible or non-purgative salts, and he therefore obtained from this experiment no proof of the correctness of his assumption. The method, however, is admittedly faulty.

I have also made an experiment with the same object as Buchheim, and although by a different and more satisfactory method, yet with practically the same results. I made 10 per cent. solutions of a number of purgative and non-purgative

TABLE SHOWING RATE OF EVAPORATION OF 10 PER CENT. SOLUTIONS OF CERTAIN CRYSTALLINE SALTS.

Inside Diameter of Tube.		1·00 cm.	1·82 cm.	1·83 cm.	1·82 cm.	1·81 cm.	1·84 cm.	1·81 cm.
Date.	Hour of Observation.	Chloride of Sodium.	Chloride of Sodium.	Acetate of Soda.	Sulphate of Soda.	Phosphate of Soda.	Sulphate of Magnesia.	Tartrate of Soda and Potash.
		cm.	cm.	cm.	cm.	cm.	cm.	cm.
Aug. 31	1.30 p.m.	17	17	17	17	17	17	17
Sept. 7	" "	16·40	16·35	16·40	16·45	16·40	16·40	16·40
" 15	" "	16·00	15·90	16·00	15·90	15·90	15·90	15·90
" 23	" "	15·60	15·45	15·60	15·45	15·50	15·50	15·45
" 27	2.10 "	15·40	15·30	15·35	15·30	15·30	15·30	15·30
	Sulphuric acid renewed.							
Oct. 5	12.35 "	15·15	15·05	15·05	14·95	15·00	15·05	14·95
" 13	1.30 "	14·90	14·80	14·85	14·75	14·70	14·75	14·70
" 21	5.15 "	14·70	14·50	14·60	14·50	14·45	14·55	14·50
Nov. 1	1.30 "	14·32	14·25	14·22	14·15	14·00	14·20	14·15
" "	2.30 <sup>2</sup> "	14·40	14·27	14·22	14·25	14·25	14·25	14·15

salines, and placed them in glass tubes of nearly equal and of perfectly uniform diameter, standing exactly vertically, and arranged in a circle round a large open beaker containing strong sulphuric acid. The whole was covered with a glass bell-jar well sealed to a ground glass plate, so that there was no

<sup>1</sup> Buchheim, *Beiträge z. Lehre v. d. Endosmose*, &c., Archiv. f. physiolog. Heilkunde, 1853, S. 217.

<sup>2</sup> These readings were made after a slight crystalline efflorescence, which had formed inside the upper empty part of the tube, had been scraped down into the solution.

communication with the outer atmosphere. The tubes were carefully graduated by lineal measurement, not according to capacity, and were filled to an equal height with the various solutions. Daily observations of the decrease of the fluids were made for over a period of two months. The room in which the tubes were placed was kept at a tolerably uniform temperature throughout the experiment. In the foregoing table I give, with one or two exceptions, every eighth observation.

A second solution of chloride of sodium, it will be observed, was placed in a tube a little narrower than the others, in order that I might be able to calculate what effect the unavoidable small variations in the diameters of the other tubes might have on the rate of evaporation.

The result of the experiment was, as can be readily seen, that evaporation proceeds with an almost equal rate in all the saline solutions, and that, therefore, in so far as the affinity of salts for water can be tested by this method, the affinity of the purgative salts is not greater than that of non-purgative salts. That a salt does, however, diminish the rate of evaporation of the water with which it is dissolved—a fact long recognised—is shown in this experiment by the rate of evaporation gradually diminishing, as the salt solution becomes more concentrated. This was not due to the sulphuric acid beginning to lose its hygroscopic action on account of its becoming saturated with moisture, for, even after the renewal of the acid, the rate of evaporation still continues to diminish.

As the method which I employed for testing the affinity of salts for water is, like Buchheim's, not without many objections, I have had recourse to another method, and, without further experiment, have availed myself of its results as found in the researches of Berthelot,<sup>1</sup> Thomsen, and others, which have recently been welded by the first of these into a new system of chemistry under the name of *Mécanique Chimique*. These chemists measure the strength of affinity between two substances by estimating the amount of heat developed when they are mixed together. Now, most of the hydrated crystalline salts when dissolved in water, instead of evolving heat, absorb it. This has long been known, and some measurements of the degree of cold

<sup>1</sup> Berthelot, *Mécanique Chimique*, Paris, 1879.

were made by Favre and Silbermann; but it is to the more recent investigators that we are indebted for more exact and reliable results. If the evolution of heat be evidence of attraction, absorption of it can hardly be otherwise regarded than as indicating repulsion; and the greater the absorption of heat the greater will be the degree of repulsion. Judged by this view of their thermo-dynamics, the purgative and less diffusible salts, owing to their absorbing a large amount of heat when dissolved in water, are those which repel water most, and in this respect the phosphate of soda acts the most energetically of all the purgative soda salts; whereas the non-purgative and less diffusible salts, as the chlorides, because they dissolve in water with a small absorption of heat, repel water the least. Instead, therefore, of there being a greater affinity between purgative or less diffusible salts and water, there is a less affinity than between water and non-purgative or more diffusible salts.

Clearly, then, the only way in which a highly indiffusible salt, as phosphate of soda, can retard the absorption of the intestinal fluid in the course of purgation, is by the presence of its slowly absorbable molecules mechanically hindering the free absorption of the molecules of the water in which it is dissolved. Molecules of both the salt and water lie in contact with the surface of the mucous membrane; those of the former, on account of their being very slowly removed by absorption, prevent the ready access of those of the latter to the absorptive cells of the membrane. In this manner, therefore the less diffusible salts can, *cæteris paribus*, purge more powerfully than those more diffusible.

I have now discussed the two properties to which I believe the greater activity of certain salts within the intestines is due, and which therefore constitute these salts cathartics, as distinguished from other allied salts, which, although not devoid of purgative action, yet do not act so powerfully as the cathartic salts proper. These two properties are bitterness and slow diffusibility, the one promoting secretion, the other impeding absorption. But whilst the possession of these properties may explain why certain salts are pre-eminently cathartic, it does not explain why other allied salts, as the alkaline chlorides and acetates, although devoid of bitterness and easily diffusible,



nevertheless excite more or less secretion when directly injected into the intestine, or purge when administered by the mouth in a sufficiently large dose. Doubtless the strongly irritant action of many of these salts, as chloride of sodium and nitrate of potassium, is sufficient to account for their purgative action ; but there are others of them, as acetate of potash, which cannot be said to be more irritant than the ordinary saline cathartics. It is therefore probable, indeed certain, that all salts, apart from their being bitter and without that they cause visible redness or inflammation of the mucous membrane, can, if sufficiently concentrated, stimulate the mucous membrane of the intestine, when brought into contact with it, and reflexly provoke the intestinal glands to action ; if for no other reason than that, on account of their concentration, they abstract water from the epithelial cells on the mucous surface, and thus, by disturbing the cell-protoplasm, affect the sensory nerve filaments, which terminate in or around the protoplasm. This very simple and purely physical effect is probably the basis of the action of every salt, if sufficiently concentrated, on living tissue, and, in this particular instance, of the purgative and non-purgative salines alike, or what might be more strictly termed, the more purgative and the less purgative. In the former the stimulant action of this property is increased by the bitterness, and the purgative effect aided by the slow diffusibility of the salt.

It is also to be remembered that the changes, by way of decomposition, which certain of the saline cathartics undergo in their passage through the alimentary canal, may materially promote their action. The splitting up of a portion of the sulphates of magnesia and soda in the intestines, with the absorption of the disengaged sulphuric acid, will probably result in the residual base uniting with the carbonic acid of the intestinal secretion to form a carbonate or bicarbonate, which, on account of its being less diffusible and less capable of absorption than the corresponding sulphate, will increase the purgative action of the salt. The return of the original acid of the salt to the intestine, as indicated in the experiments of Series D., taking place, as it evidently does (Experiments CXVI. and CXVII., Series H.), to a small extent in proportion to the

amount of the base still within the intestine, will not entirely break up the carbonate formed. Beyond this, I do not believe, and therefore in opposition to Headland, that the absorption and after excretion of the acid of the salt affects its purgative action. The absorbed portion of the salt in the process of its excretion probably plays no part in stimulating the intestinal secretion. My reasons for holding this opinion I have already stated.<sup>1</sup>

In considering the properties which render certain salts cathartics, I have assumed that the salts possess no *specific* stimulating action on the enteric glands, as do those of nearly all the heavier metals. In this I may not be altogether accurate, for whilst it may be held as almost indisputably proved that the salts of soda have no such action, otherwise there would be evidence of it when they are injected in purgative doses into the blood, yet the salts of potash or magnesia may possess it in some degree, as, owing to their highly poisonous effect when injected directly into the circulation, it is impossible to inject them in a quantity approaching a purgative dose. If they do possess a specific purgative action, it must be to a very limited extent, for the still more poisonous salts of the heavier metals rarely fail to excite purgation when introduced into the blood in less even than a lethal dose.

In treating of the nature or mode of production of the purgative secretion by the salt, I have omitted all references to the effect being produced by the action of the salt on any portion of the body outside the nervous and glandular structures of the intestine, for the simple reason that the application of the salt *per se* to any other portion of the body, either by intravenous or by subcutaneous injection, completely fails to cause purgation. I do not, however, deny the possibility of the local intestinal action being controlled by some nervous influence originating in a separate portion of the organism, and altogether independent of the action of the salt. The influence may be such as either to increase the purgative action or to lessen it, and these results it may effect by altering the rate either of secretion or of absorption in the intestine. For example, I have shown in Experiments XLI. and XLII. that irritation by

<sup>1</sup> Vol. xvi. p. 587.

ligatures of a portion of the intestine distant from the part in which the salt is acting, diminishes the purgative action by promoting secretion. This effect probably does not take place through a purely intra-intestinal nerve mechanism; a much more extended nerve-tract is in all probability involved, consisting at least of the mesenteric nerves and ganglia. Also, according to H. C. Wood, division of the vagi in the neck prevents purgative action by arresting the intestinal secretion. I have not, however, succeeded in observing this inhibitory effect when a saline cathartic was employed.<sup>1</sup>

The intestinal secretion is also affected by subcutaneous irritation in the region of the abdomen (Series of Experiments F.), and there can hardly be any doubt that either in a normal condition, or when stimulated by a purgative, it may be largely affected by many nervous influences, emotional and otherwise.

*V. The Action of the Salt on the Peristaltic Movements of the Intestines.*

The determining whether the salt stimulates these movements is of somewhat less interest, since I have in the course of this investigation removed all uncertainty as to the salt exciting secretion. In the historical sketch at the commencement of this paper, I have remarked how that the German pharmacologists are inclined to regard such a stimulation as the principal, or at least an important, cause of the catharsis. The experiments of Aubert, and Buchheim and Wagner, but more particularly those of Thiry and Radziejewski, form the basis of this belief, without that any of their experiments offer direct proof of increased peristalsis. Legros and Onimus,<sup>2</sup> and Van Braam Houckgeest,<sup>3</sup> have, however, by direct experiment, demonstrated that a saline

<sup>1</sup> Since writing the earlier part of this paper, in which Wood's experiments are shortly commented on, I have had the opportunity of perusing a detailed account of his experiments in the *American Journal of Medical Sciences*, vol. lx. p. 75. I find that he does not attribute the effect of the division of the vagi to any direct influence exerted by these nerves on the secretion of the intestines, but to the accumulation of carbonic acid in the blood and congestion of the portal circulation, caused by the division of the vagi. The shorter account of his experiments, which I had previously read, does not give the same explanation of the action of the divided vagi, and I have, therefore, felt it incumbent to make this correction.

<sup>2</sup> *Op. cit.*

<sup>3</sup> *Op. cit.*

purgative, on the contrary, does not stimulate the movements of the intestines. The fitness of the methods employed by the latter observers, and the care with which their experiments were conducted, command acquiescence in their conclusions. In all those of my own experiments in which the intestines were exposed, although I frequently made observation of the peristalsis, I could never on any occasion satisfy myself that it was increased during the action of the salt. I observed, however, in an experiment specially instituted, that moderate distension of the intestine with what may be regarded as the blandest of all ordinary fluids, a  $\frac{3}{4}$  per cent. solution of common salt of the same temperature as the body, excited regular contractions of the intestinal wall of the distended loop, which ceased immediately on removal of the pressure. Although, therefore, the salt does not directly stimulate the peristaltic movements of the intestines, yet it is highly probable that it indirectly does so by distending the intestines with the abundantly poured-out secretion. Increased peristalsis may, accordingly, be a subsidiary factor in the action of a saline cathartic, but certainly it is not an essential factor.

#### B. THE EFFECT OF THE SALINE CATHARTIC ON OTHER PARTS OF THE BODY THAN THE ALIMENTARY SYSTEM.

As the action of the salt on the circulation and urinary secretion has already been fully discussed to the extent my experiments warrant, and under separate chapters, I shall do no more here than briefly recapitulate the conclusions already arrived at.

##### I. *The Effect on the Blood and Circulation.*

The salt, by exciting a profuse intestinal secretion, removes, under all circumstances, a large amount of fluid from the blood. If the salt is administered in very dilute solution, the water of the solution by absorption replaces the water of the blood removed by secretion. But if the solution of the salt is concentrated, little or no replacement occurs, and the total bulk of the blood in the body becomes greatly diminished. This concentration of the blood lasts, however, only for one or two hours, because by the end of that time the blood recoups itself from the fluids of the tissues. The action, therefore, of a diluted dose of a saline cathartic on the blood, and secondarily on the

tissue fluids, is considerably different from that of a concentrated dose. The use of the latter would seem to be strongly indicated in the treatment of many forms of dropsy where it is desired to obtain a rapid and powerful reduction of the dropsical fluid. I have already made such an application of it, and with the happiest results.<sup>1</sup>

The profuse intestinal secretion removes with it a large amount of the salts of the blood, but apparently very little of the organic matter, and this will occur whether the solution of the salt administered be concentrated or dilute, although the more free absorption in the latter case will restore a certain proportion of the secreted salts. But if the blood thus loses a portion of its natural salts, its inorganic matter is increased by the presence of the absorbed salt, more of the acid of which than of the base would appear to enter the blood.

A saline cathartic causes, therefore, a partial depletion of the blood—partial, in so far as it only removes a portion of the inorganic constituents, and more or less of the water, along with a mere trace of the dissolved and unformed organic matter, and, unlike an ordinary surgical depletion, removes none of the formed constituents or corpuscular element.

After the purgative action of the salt is at an end, at any rate in so far as the blood is concerned, the diuretic action of the absorbed salt, which is now established, or rather permitted to become apparent, causes a second concentration of the blood, considerably less in degree than the first, but continuing throughout a much longer period. This concentration is probably not characteristic of purgative salts solely, but is also to be observed after the administration of the ordinary diuretic salts.

The presence of the salt in the blood, and its contact with the various tissues and organs, does not, so far as I have been able to make out, affect to any appreciable extent the metabolism of the body.

As regards the effect of the salt on the circulation of the blood, the salt appears to increase the blood-pressure by causing a contraction of the smaller arteries and the capillaries, merely, as I believe, on account of its mildly stimulating the tunica intima of the vessels as it circulates with the blood.

<sup>1</sup> *Lancet*, April 21, 1883.

*II. The Effect on the Urinary Secretion.*

The immediate effect of the administration of the salt is, as in the case of the blood, somewhat dependent on the degree of concentration of the blood. If the solution of the salt is concentrated, and the blood, therefore, rendered also concentrated, the urinary secretion is at first diminished. If, on the other hand, the solution is very dilute, the secretion may be increased by the blood and kidneys removing the excess of water from the intestine. In both cases, after some hours, and, probably after the salt has ceased to exert any active purgative effect, the secretion increases, and a veritable diuresis would appear to be established and to continue for nearly a day.

The salt produces no material alteration of the composition of the urine, beyond that due to its own presence. In the case of sulphate of magnesia, I have proved that much more of the acid of the salt than of the base appears in the urine. This may also occur to a certain extent with other purgative salts.

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These are the main results of this lengthy investigation of the physiological action of saline cathartics; and although they have mainly been obtained from experiments with sulphate of soda, and in a few instances sulphate of magnesia, yet these salts are sufficiently typical of the whole group of saline cathartics as to perfectly justify the belief that, had other members of the group been employed, similar results would have been procured. I have to a certain extent pointed out the differences in manner of action which distinguish some of them. They all tolerably closely agree in the ultimate effect they have on the alimentary canal and the body generally. They cause no irritation or inflammation of the canal; stimulate but in the smallest degree the secretion of the more important digestive juices, as the gastric, the pancreatic, and the biliary; have under ordinary circumstances little action on the blood; and mainly act by increasing the intestinal secretion and by hindering the absorption of the intestinal fluid. Their purgative action is, therefore, extremely simple. They sweep out the contents of the alimentary canal with the least possible disturbance of the digestive system and of the other systems of the organism. Few

other purgatives, if any, have so simple an action. The value, therefore, which has long been assigned to them in the treatment of the occasional disturbances of digestion, to which almost every one is at times subject, and where the indication seems to be to empty the canal "*cito, tuto, et jucunde*," is quite justified by the results of this investigation.

This is a long research and has involved much labour. It was commenced in the laboratory of the Pharmacological Institute at Strassburg under the superintendence of Professor Schmiedeberg, where most of the experiments of the A. series were made. I have already acknowledged my indebtedness to Professor Schmiedeberg for his valuable and kindly help in the carrying out of these experiments. As I did not altogether feel assured of the correctness of the theory of the mode of action of saline cathartics, to which the results of these experiments seemed to lead, I reopened the investigation, after my return to Edinburgh, in the laboratory of the Pharmacological Department of this University, where nearly all the experiments of the remaining series were conducted. Here I have received valuable assistance from many friends; and for such, more than to any other, do I feel grateful to Professor Thomas R. Fraser, but for whose kindness and indulgence the research could not have been continued. I have also to warmly thank Dr J. R. Logan, Dr Hosack Fraser, Dr. J. O. Liddell, Dr. J. H. Balfour, and Mr. James Stewart, who on certain occasions, and sometimes at considerable inconvenience to themselves, have kindly given me their help.

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#### SUMMARY OF THE RESULTS OF THIS INVESTIGATION.

1. A saline purgative always excites more or less secretion from the alimentary canal, depending on the amount of the salt and the strength of its solution, and varying with the nature of the salt.
2. The excito-secretory action of the salt is probably due to the bitterness as well as to the irritant and specific properties of the salt, and not to osmosis.
3. The low diffusibility of the salt impedes the absorption of the secreted fluid.

4. Between stimulated secretion on the one hand, and impeded absorption on the other, there is an accumulation of fluid in the canal.

5. The accumulated fluid, partly from ordinary dynamical laws, partly from a gentle stimulation of the peristaltic movements excited by distension, reaches the rectum and produces purgation.

6. Purgation will not ensue if water be withheld from the diet for one or two days previous to the administration of the salt in a concentrated form.

7. The absence of purgation is not due to the want of water in the alimentary canal, but to its deficiency in the blood.

8. Under ordinary conditions, with an unrestricted supply of water, the maximal amount of fluid accumulated within the canal corresponds very nearly to the quantity of water required to form a 5 or 6 per cent. solution of the amount of salt administered.

9. If, therefore, a solution of this strength be given, it does not increase in bulk.

10. If a solution of greater strength be administered, it rapidly increases in volume until the maximum is attained. This it accomplishes in the case of a 20 per cent. solution in from one to one and a half hours.

11. After the maximum has been reached, the fluid begins gradually and slowly to diminish in quantity.

12. *Cæteris paribus*, the weaker, or in other words, the more voluminous the solution of the salt administered is, the more quickly is the maximum within the canal reached; and accordingly purgation follows with greater rapidity.

13. Unless the solution of the salt is more concentrated than 10 per cent. it excites little or no secretion in the stomach.

14. The salt is absorbed with extreme slowness by the stomach of the cat.

15. The salt excites an active secretion in the intestines, and probably for the most part in the small intestine, all portions of this viscus being capable of yielding the secretion in almost equal quantities.

16. The bile and pancreatic juice participate but very little in the secretion.

17. The secretion is probably a true *succus entericus*, re-



sembling the secretion obtained by Moreau after division of the mesenteric nerves.

18. The secretion is promoted by local irritation of the intestine, as by ligatures, but only in the immediate vicinity of the irritation.

19. Absorption by the intestine generally is reflexly stimulated by such irritation (the effect of numerous ligatures applied at points remote from the seat of the injected salt being to diminish the amount of purgative fluid by accelerated absorption).

20. If the salt solution be injected directly into the small intestine, the stronger within certain limits the solution is, the greater will be the accumulation of fluid within the intestine.

21. This difference is not observed when the salt is administered *per os*, as the strong solution becomes diluted in the stomach and duodenum before passing into the intestine generally,

22. The difference is due to the local action of the salt on the mucous membrane, and probably more to an impeded absorption than to a stimulated secretion.

23. When the salt is administered in the usual manner, it appears, in the case of the sulphate of magnesia and sulphate of soda, to become split up in the small intestine, the acid being more rapidly absorbed than the base.

24. A portion of the absorbed acid shortly afterwards returns to the intestines.

25. After the maximum of excretion of the acid has been reached, the salt begins very slowly and gradually to disappear by absorption, which is checked only by the occurrence of purgation.

26. During the alternations of absorption and secretion of the acid, it is the salt left within the intestine which excites secretion, the absorbed and excreted acid exerting no such action whilst in the blood, or during the process of its excretion, as Headland believed.

27. The salt does not purge when injected into the blood, and excites no intestinal secretion.

28. Nor does it purge, when injected subcutaneously, unless in virtue of its causing local irritation of the abdominal subcutaneous tissue, which acts reflexly on the intestines, dilating

their blood-vessels, and perhaps stimulating their muscular movements.

29. The sulphate of soda exhibits no poisonous action when injected into the circulation.

30. The sulphate of magnesia is, on the other hand, powerfully toxic when so injected, paralysing first the respiration and afterwards the heart, and abolishing sensation or paralysing the sensory-motor reflex centres.

31. Both salts, when administered in the usual manner, produce a gradual but well-marked increase in the tension of the pulse.

32. According as the salt-solution within the intestine increases in amount, there occurs a corresponding diminution of the fluids of the blood.

33. The blood recoups itself in a short time by absorbing from the tissues a nearly equal quantity of their fluids.

34. The salt, after some hours, causes diuresis, and with it a second concentration of the blood, which continues so long as the diuresis is active.

35. As the intestinal secretion excited by the salt contains a very small proportion of organic matter as compared with the inorganic matter, the purgative removes more of the latter than the former from the blood. In certain cases a large quantity of the salts of the blood is thus evacuated.

36. The amount of the normal constituents of the urine is not affected by the salt.

37. After the administration of sulphate of magnesia much more of the acid than of the base is excreted in the urine.

38. The salt has no specific action in lowering the internal temperature of the body, or has it only to a very small extent.

39. It reduces, however, the absolute amount of heat in the body.

SOME REMARKS ON THE ANATOMY AND PHYSIOLOGY OF THE URINARY BLADDER, AND OF THE SPHINCTERS OF THE RECTUM. BY F. LE GROS CLARK, F.R.S.

THE usually accepted views on the physiology of micturition have always seemed to me unsatisfactory. The subject is a perplexing one, much more so than is apparent until it is carefully investigated; and it is only after much consideration that I venture to express dissent from the opinions generally received, and my reasons for adopting others in their stead. The value of a correct view is, in this instance, enhanced by the light thereby thrown upon many obscure pathological conditions, which, in their turn, serve to illustrate the physiology allied with them. The chief points I propose to discuss are:—

1. How the urine is expelled from the bladder.
2. Under the influence of which of the nerve-centres the expelling force acts.
3. How the urine is retained in the bladder under different circumstances.
4. The relation of various pathological conditions to the physiological explanation offered.

I may remark that I once wrote a paper on this subject, which was published in the *London Medical Gazette* for June 18, 1836. It attracted little attention, because it contained doctrines which were regarded as crude and erroneous; for such was the view then taken by most physiologists of Marshall Hall's great discovery. Reflex physiology was just struggling into life, amid all the detraction and opposition which so commonly herald and accompany the announcement of new views which revolutionise previously established opinions. I have only recently disinterred this forgotten production of my youth; and this is the explanation of my renewing the inquiry after so long an interval.

*Anatomy.*—The arrangement of the fibres which constitute the *muscular coat* of the bladder is such as to enable it to contract uniformly on its contents. When the organ is elongated by distension, the longitudinal fibres shorten this diameter; and

when partially emptied, the circular and oblique fibres are probably more actively engaged. The circular bands increase in number as they approach the neck of the bladder, where they form a more compact layer, especially when the bladder is contracted.

The position and relations of the *prostate gland* are such that it probably exercises but little if any influence on the passage of the urine. Circular muscular fibres constitute a considerable element in the vesical half of its structure, and therefore behind the opening of the ejaculatory duct. My friend and former pupil, Mr. Anderson, conjectures that these fibres have for their function the occlusion of the urethra at this part during emission of the semen; and no doubt they express the secretion of the gland at the same time and in the same required direction, so as to bring both secretions within the grasp of the ejaculator muscles. These prostatic fibres can scarcely be credited with any agency associated with micturition, as they have no homologue in the female. The relation of the levator ani to the prostate might suggest that this muscle is capable of compressing it, and of thus aiding in closing the urethra. But it is more than doubtful whether these fibres in any degree envelop the gland, as usually described by anatomists, after Santorini; they really pass from its sides on to the rectum.

The *membranous urethra* is composed of elastic and erectile tissues, with circular muscular fibres. The female urethra corresponds, in position and structure, with this portion in the male; but its elastic fibres, as well as its erectile tissue and the muscular fibres which encircle it, are more developed in the former than in the latter. There is one muscle common to both sexes, viz., the *compressor urethræ*, which surrounds the urethra whilst beneath the arch of the pubes and between the layers of the triangular ligament. It is needless to say that, although the accelerator urinæ and erector penis have their homologues in the female, their function in this sex has no relation to the act of micturition.

The *vesical nerves*, derived from the lower part of the hypogastric plexus, consist of spinal nerves, intermingled with others derived from the lumbar and sacral ganglia of the sympathetic.

Impressed as I was with the prevailing belief that the emptying of the bladder is a purely reflex act, I proceeded to examine

the correctness of this view in the following way. Given, a bladder containing a small quantity, say three or four ounces, of water, and no desire to expel it, how is micturition accomplished? The ability cannot be doubted; and the act is clearly voluntary. The exercise of the will must be either in active expulsion, or in suspension of obstructive control. But this difficulty has been met by the assertion that contraction of the abdominal muscles is the initiation of the act. I therefore tested this statement by a careful examination of the condition of these muscles during micturition, in various states of the bladder as regards the quantity of urine it contained, with the result of satisfying myself that there is no evidence to support this supposition. Moreover, no voluntary effort of the abdominal muscles suffices, by itself, to stimulate the bladder to respond, when its action is suspended emotionally, as by nervous apprehension or shame,—a condition to which this organ seems peculiarly susceptible, though not singularly so; for strong emotion will paralyse other muscles which are more directly subject to the will; or, more properly speaking, which are less generally influenced by the excito-motor centre. No doubt the abdominal muscles may be employed voluntarily, and usually are so when a desire exists to accelerate the passage of the urine; and also immediately preceding the final ejaculatory act by the perineal muscles: but my contention is that, unless thus called into action, these muscles are perfectly passive. Any contraction of the abdominal wall, which would suffice to initiate the act of micturition when the bladder contains only a small quantity of water, must, in a thin person, be obvious to the touch. But experiment may satisfy anyone that voluntary contraction of the abdominal wall, so far from facilitating the commencement of the act, really obstructs and delays it; the effort is sensibly of a different nature. In defæcation, likewise, though the abdominal muscles very generally take an active part, their intervention is not essential; the peristaltic movement of the intestine can by itself suffice, as proved in diarrhœa, or, still more manifestly so, where voluntary power is suspended by disease or injury.

If, however, under the conditions mentioned and excluding the abdominal muscles from any necessary participation, it be still maintained that the muscular coat of the bladder is not

under the direct government of the will, the only alternative is to assume that the bladder needs and possesses a strong sphincter, which is in constant reflex action, controlling the outlet; that the will is capable of inhibiting this reflex action; and that such control, when exercised, simultaneously excites the reflex contraction of the bladder. But can such a complicated assumption be regarded as consistent with accepted physiological doctrine and observation? I believe not; even if the assumed need for the presence of such a sphincter were admissible, which I shall endeavour to show is not the case. In thus expressing myself I do not intend to deny that the bladder may and does act under reflex influence. All muscles are subject, more or less, to this agency, and probably the bladder is peculiarly so. What I contend for is that the will has direct power in determining its contraction. But I hope to illustrate this branch of the subject further on.

I now proceed to inquire how the bladder retains its contents under different circumstances. In considering this question in relation to both sexes, it is necessary to exclude the prostate gland and the urethra in front of its membranous division, which is, in the male, the homologue of the female urethra.

Assuming, for the sake of argument, that the bladder requires a restraining sphincter, what muscular arrangement exists by which the expulsion of the urine may be controlled? If the annular fibres around the neck of the bladder be credited with this function, then the inhibition of their action must be voluntary and synchronous with the reflex contraction of the muscular coat of the bladder generally, with which they are continuous and intermingled, as well as identical in structure. I have already stated my reasons for rejecting this supposition. Moreover, these circular fibres are not aggregated in the form in which we should expect a sphincter muscle to present itself when the bladder is distended; but they are so arranged as to constitute a detractor muscle when and where it is most required, viz., when the bladder is nearly emptied, and the urine gravitates towards its neck; just, in fact, as the mixed annular fibres at the lower part of the rectum act in expelling its contents, after being expanded by the pressure from behind.

I formerly thought (as expressed in the paper to which I have

alluded) that the accelerator urinæ might act as a sphincter: but, except under special circumstances, I do not now think this is the case; for the retaining power of the bladder seems to be very little interfered with when the urethra is laid open behind the bulb: moreover, its homologue in the female can have no such action. The position of the compressor urethræ is such as to qualify it to close the opening from the bladder into the urethra: but would the inhibition of its action suffice to liberate that opening, and to stimulate the muscular coat to reflex action when the bladder contains only a small quantity of water? I apprehend that such an explanation could scarcely be entertained seriously.

In considering the retaining capability of the bladder we have to take into account the hydrostatic condition of the organ, the elasticity of its outlet and excretory duct, and the muscular apparatus in connection therewith. Of these I consider the first or passive resistance to evacuation as by far the most potential. For all practical purposes we may regard the bladder as a spherical reservoir with but one outlet, as the ureters are really closed against any retrogression of the urine. If the ratios of the pressure between this large reservoir and the small cylindrical tube with which it is connected are as the respective areas of the tube and cistern, the collapse of the urethral wall could easily counterbalance a very considerable hydrostatic pressure, together with that which is exerted by the elastic resiliency of the distended bladder. My friend, Dr. Stone, whom I have consulted on this point, informs me that he estimates the tension on the internal area of the bladder to be about 560 times that on the vesical outlet. A certain allowance must also be made for the friction consequent on the passage of the fluid through a long and tortuous channel. The absence of this obstructive agency in the female ought to be, and I presume is, attended with increased force in the current of the fluid when it leaves the canal. Experimentally we know that this passive retaining attribute of the bladder exists. After death we often find a considerable quantity of urine in the human bladder, and also in the bladders of slaughtered animals; in neither case is it expelled, as it would be if the retention were dependent on muscular action. I have often seen the fluid contents of the stomach discharged

by the mouth, immediately the diaphragm has relaxed its hold on the cardia in death, and the elastic pressure of a distended abdomen has been therefore unopposed. If we superadd to this passive resistance in the bladder that offered by the elastic and muscular texture of the urethra, which is more developed in the female than in the membranous division of the male canal, we have, in my opinion, a satisfactory explanation of the normal retention of the urine.

But, as we are conscious of the desire to micturate, so are we sensible of being able to exercise an effort to restrain the action of the bladder. What is this? In the first place, I believe it to be a voluntary inhibition of the contraction of the bladder when stimulated by a centripetal exciting cause, whether that be an appeal to the sensorium or to the spinal centre. Sometimes in the contest, resulting from over-distension or morbid irritability of bladder, volition is compelled to yield. But beyond this passive restraint there is the compressor urethræ muscle, which, though feeble in itself, acquires considerable importance from its position at the outlet and its relation to the urethra. If the hydrostatic pressure on the vesical outlet is relatively so inconsiderable, it is not unreasonable to believe that the action of this small muscle sensibly augments the retaining power of the bladder. Probably this function is exercised only when the bladder is distended, and as a reflex consequence of such distension; but it may, like that of the cutaneous sphincter and in similar circumstances, be exerted under the direction of the will. Thus, I infer that the effort by which we control the desire to micturate is due to the restraint exercised by the will, and to the action of the compressor urethræ muscle, which may be regarded as analogous, in its relations and functions, to its more powerful neighbour, the cutaneous sphincter of the rectum. Both are called into action in the erect posture, and during any exertion in which the abdominal muscles are usually engaged, as in coughing. And I may here remark that, if my data and argument be not impugned, it is impossible to suggest any sufficient check to the evacuation of the bladder, when sensibly stimulated by distension, unless it be, at any rate chiefly, that of direct voluntary control over the expulsive force; and if such exercise of volition be admitted, it seems to me that the direct predominant agency of the will in



determining the expulsive action cannot be denied, *i.e.*, so long as sensibility is unimpaired.

I will now say a few words respecting other parts of the male urethra. As regards the prostate I do not think, as I have already remarked, that the healthy gland exercises any influence on the passage of the urine through it: but in front of the membranous urethra the presence of a powerful muscle, the *accelerator urinæ*, commanding the passage, requires notice. Its special function in micturition is to clear the passage, in front of it, of the water that would otherwise dribble away. This is accomplished, not only by compression of the bulb, but also by producing, in concert with the muscles of the crura, a momentary distension of the erectile tissue, which passes forwards as a wave towards the extremity of the canal. But the accelerator muscle has another function which must be necessarily absent in the female, *viz.*, that of suddenly arresting the flow of water during micturition. I believe, from observation of such cases, that spasm of this muscle is the usual obstruction in what is termed spasmodic stricture; and that otherwise this form of obstruction (except it be also in the membranous urethra and due to the compressor muscle), is very much of a myth, which has often served the purpose of an excuse for clumsy catheterism. Further, I do not doubt that, in some instances, the accelerator muscle, when acting under reflex influence, is capable of resisting considerable pressure from behind. To compensate for this, and for the resistance from friction in the male canal, both the elastic and muscular elements of the female urethra are more developed than in the membranous urethra of the other sex. Even the erectile tissue, especially in the female urethra, may aid, when the compressor is in action, in closing the canal. In the erect posture and during active exertion there is a general tonic contraction, not only of the sphincters, but also of the levator ani and other muscles commanding the pelvic outlet. A similar arrangement may be observed, even in ordinary inspiration, when the body is erect: the abdominal muscles are no longer the passive wall that they are in the recumbent posture, for the abdominal breathing is not then so pure and simple; but it is limited, for an obvious reason, by the tonic action of the surrounding muscles.

I may here notice an objection which will naturally be raised

to my argument, and that is the anatomical distinction between the voluntary and involuntary muscles. But are we to reject the proposition that the muscular coat of the bladder is under the direct influence of the will because it is composed of unstriped fibres? If so, why not deny involuntary activity to the striped fibres of the heart, pharynx, and œsophagus, or voluntary action to the ciliary muscle? This physiological distinction on anatomical grounds is only arbitrary, and is unsupported by any physiological necessity of alliance between structure and function. Why some muscles are striped and others unstriped is an unsolved problem, which may hereafter be shown to characterise differentially their functional activity, or to have more relation to the distribution of the cyclo-ganglionic nerves than to any special function associated with the cerebro-spinal system; or to be totally unconnected with either. I may further observe that most spinal-reflex acts are more or less subject to the control of the will. Such is the case with the action of the sphincters, the larynx, the diaphragm. The contraction of the pharynx and œsophagus seems to be purely reflex, though the initiation of swallowing is voluntary; and these specially reflex and involuntary muscles belong to the striped class.

In studying the reflex phenomena one cannot fail to be struck with the remarkable development of this function in early life. Reflex movements, under appropriate stimulants, are most manifest where voluntary influence is most deficient; and the converse is also true. The will has to be educated and habit must be acquired before volition supersedes reflex action. These remarks apply to the act of micturition as well as to the action of muscles generally. Indeed, until education and habit have asserted their sway, it would seem that the evacuation of the bladder is a purely reflex act. This is remarkably exemplified in the case of anencephalous infants, in which not only are respiration and deglutition performed naturally, and the limbs moved when excited, but the sphincters are active, and the urine and fæces are voided. A case of this kind is narrated by Sir William Lawrence in the fifth volume of the *Medico-Chirurgical Transactions*; and this account preceded the discoveries of Dr. Marshall Hall, who quotes similar instances given by Lallemand and Ollivier. The fact is, that all the actions essential to the

maintenance of earliest life, including suction, are reflex : and is it not the fact that in old age, as the energy of the will (in a physiological sense) abates, reflex influence again becomes relatively more active, as manifested especially in morbid conditions of the frame? The controlling power of the will is evoked by education and confirmed by habit; the primitive condition offering an interesting subject of study in contrast with that which is presented in after-life, when volition is suspended by cerebral or spinal injury. The influence of habit is exemplified by control of the bladder during sleep, by the phenomena of somnambulism, and in various other ways known to physiologists. In like manner, numberless voluntary acts become purely automatic by habit; they are performed unconsciously, and even when they would be checked if the will were not slumbering. Still more striking instances are on record of such habitual movements occurring when the brain is incapable of taking cognisance of external objects. The power of association also is manifested in the desire to urinate which is excited by washing the hands, retiring to bed, &c. Similar instances illustrate the influence of habit and education in domestic animals. The puppy will pass water whenever his bladder is distended, and wherever he may be; but the adult and trained dog will remain for hours indoors without urinating, although he will do so repeatedly and at short intervals when running free. The horse, again, will delay to stale on a long journey until he reaches his own stable. But it is unnecessary to pursue this subject further.

In studying the Pathology of micturition we have to consider those conditions in which the bladder either expels or retains abnormally its contents; in other words, incontinence and retention: and each of these states may be the result of disease or injury.

*Incontinence* may occur at any age, and is due to various causes. In children it is not uncommon during sleep, after education has been confirmed by the habit of retention at other times. Various remote causes may operate in exciting the act under these circumstances; but the explanation is that the centripetal stimulus is abnormally strong, or the habit of control is too feeble to resist it. At any period of life an irritable or inflamed state of the

vesical mucous membrane, the presence of an irritating body in the bladder, or an unhealthy condition of the urine, or even strong emotion, will produce the same result. Incontinence in hysteria is infrequent, but may be referred to a capricious or enfeebled exercise of the will. In old age I believe the explanation of incontinence to be, in most cases, the simple overflow of a distended bladder. The supposition that the urine passes directly from the ureters through a collapsed and atonic bladder is forbidden by the operation of the hydrostatic law already referred to, even assuming that all active obstruction is removed. In such cases the urine may be retained whilst the patient is in the recumbent posture or at rest; but exercise in the erect posture is accompanied by its discharge. This is attributable, in great measure, to the pressure of the abdominal muscles on the bladder, together with the stimulus to urinate consequent thereon. Females, in like manner, are occasionally subject to incontinence during pregnancy, when coughing or exerting themselves; the same explanation applies in this instance, as also in some convulsive affections besides hysteria, such as epilepsy and chorea, in which the urine is occasionally expelled during the attack. In the aged, however, there is sometimes, indeed not infrequently, a morbid sensibility of the mucous membrane of the bladder, which, in concert with enfeebled power of control and relatively augmented reflex activity, produces incontinence.

The causes of *retention* of urine may be mechanical or physiological. The former class of cases present themselves in the form of an enlarged prostate, diseased growth in the bladder, the presence of a stone or clot of blood, stricture, &c. But these may be dismissed with the remark that hypertrophy of the vesical muscular fibres, so frequent in some of these cases, is a condition which is common to both voluntary and involuntary muscles when stimulated to increased action; and therefore this muscular growth does not affect the physiological question under discussion. Retention in old age is usually the consequence of defective voluntary control over an enfeebled bladder. But this condition, as already remarked, may be associated with morbid irritability of the mucous membrane, exciting occasional reflex contraction, and incontinence as a consequence. Simple dribbling of the water in retention, which is much more

frequent, is the result of pressure from distension gradually overcoming the resistance of impaired textures in front. In such cases the habit of retention is progressive without the knowledge of the patient, until at length incontinence reveals the truth. Hysterical retention, or what is so called, is more often met with than incontinence. It is a singular affection, for the bladder sometimes becomes enormously distended before relief is sought, or even complaint made. Some such cases are referable to a morbid mental or moral condition, which, if yielded to by the medical attendant, entails his constant interference. When genuine, the only explanation I can offer is that the exercise of the will is perverted or in abeyance, and that the mucous membrane is in a state of morbid insensibility, as manifested by the indifference to the distension; the consequence being that the peripheral extremities of the afferent nerves fail to convey an impression sufficiently lively to induce contraction of the bladder. A temporary incapacity to pass water sometimes occurs in nervous persons, when in the presence of strangers. This is evidently a suspension of the power of exercising the will under mental emotion, and appears to confirm the view that the beginning of the act is under direct voluntary control. Certainly in this condition no contraction, however active, of the abnominal muscles avails to stimulate into action the muscular coat of the bladder. I cannot believe that any reflex obstruction at the neck of the bladder or in its vicinity can account for this peculiar condition; for the victim of it is incapable of even making an effort to relieve himself.

Incontinence or retention may result from injury. Where the former is the case (except in excessive distension) the lesion must involve the restraining neck of the bladder, as, for example, in dilatation or section of the female urethra for extraction of a stone. The urethra in front of the neck may be largely dilated without necessarily entailing incontinence. The same is the case, apparently, in the male urethra: for Mr. Cock informs me that he has had at least two cases in which, after his operation of puncturing the front of the prostate, the patients were enabled to retain their water naturally, the urethra in front of the perineal outlet being permanently obliterated. Probably in these instances the compressor muscle may have regained its command behind the

artificial opening, though this need not be regarded as essential. In cases of extravasation of urine, treated by free incision, when a catheter is not employed to mar the healing process, I believe it will be found that the urine always escapes into the poultice at intervals and not by continued dribbling; unless indeed the bladder becomes distended, which very rarely occurs.

Retention is a consequence of lesion of the brain or spinal cord, either suspending all voluntary power or cutting it off from the bladder. In compression of the brain the retention is more or less positive in accordance with the intensity of the coma; in compression of the cord, as from fracture, where the paraplegia is complete, the bladder is absolutely inactive. This latter condition seems suggestive of the entire insusceptibility of the bladder to reflex influence when cut off from the source of sensation and volition; but Mr. Savory has directed attention to the fact that the violence and shock inflicted by crushing the cord has the effect of diminishing its reflex sensibility below the injury.<sup>1</sup> Indeed, this observation is confirmed by other experiments in such cases, designed to test that susceptibility in the lower limbs: the response is feeble, and becomes increasingly so as life wanes, though the functions of the brain may retain their activity in parts above the seat of injury. It is said that the bladder, in cases of compressed cord, will sometimes expel the urine by reflex action. I cannot say I have ever seen this exemplified. Occasionally a full bladder may, in the act of coughing, eject some of its contents, when the injury does not involve the nervous supply to the abdominal muscles. It is also said that a dog has urinated naturally after section of the cord in the dorsal region. I can believe that, the cord being divided by a clean cut and without further shock, the reflex nerve-force supplied to the bladder might suffice to excite an apparently natural ejection of the urine: but I suspect this was an exceptionally successful experiment.

I will finish with two or three supplementary observations, before I proceed to make some remarks on the sphincters of the rectum.

The sensation of a desire to pass water is an appeal to the sensorium; and this, as I believe, implies an act of volition in

<sup>1</sup> *St. Bartholomew's Hospital Reports.*

the effort that follows. The necessity for this appeal, if the consequent act be purely reflex, is not apparent, except it be as a warning to the will to restrain it. I apprehend that the condition of the muscular coat of the bladder, as regards its innervation, resembles that of the ordinary voluntary muscles. The muscular sense conveys information to the sensorium on which the will acts; in like manner the sensation of a desire to micturate is the centripetal appeal to the sensory ganglia of the brain, which excites the agency of the will; and the allied motor ganglia supply the force which is conveyed through the efferent nerves. But in each case the same muscles are also under spinal reflex influence; yet this does not necessarily imply the existence of two independent circles. On the contrary, there is reason to believe, on anatomical as well as physiological grounds, that the nerve-fibres, both afferent and efferent, communicate with the grey matter of the cord, conveying impressions to it, and receiving energy from it, in their progress to and from the brain.

The urine is retained longer, without distress, in the recumbent than in the erect posture. This difference may be due partly to the absence of tension of the parietes of the abdomen and pelvic outlet when the body is in a state of repose, but chiefly to the gravitation of the urine, and consequent pressure on the sensitive neck of the bladder in the upright position. The desire to pass water, and the commencing effort, are accompanied by a sensation as if there were some relaxation of the outlet. This sensation is referable to the same cause, namely, pressure on the urethral outlet overcoming the passive resistance there. When the act of urinating is begun, the will seems to have but feeble power in controlling its completion, except, as I have already remarked, that the outflow may be suddenly arrested in the male by the accelerator muscle. This lack of restraining power is, probably, the effect of habit in great measure; but, the passive obstruction having been overcome, the suspension of the act, without positive control, would naturally be difficult. In women the retaining power is usually greater than in men, and the female bladder is somewhat more capacious; but both these circumstances are probably the natural consequence of habit fostered by necessity.

*Some Remarks on the Sphincters of the Rectum.*

The cutaneous sphincter, which may be described as horizontal, though not strictly so, is rounded anteriorly in the female, but pointed in the male where it is attached to the accelerator urinæ, in harmony with which it acts. It is chiefly a voluntary muscle, but acts also, more or less, under spinal reflex influence, such action varying in degree according to the position, activity or rest of the body.

The intestinal or annular sphincter is in constant and vigorous action, under the command of the spinal reflex centre; it is the true guardian of the outlet, being very little under the influence of the will.

These opinions are founded on the following observations and facts:—The cutaneous sphincter resembles, in every respect, the voluntary muscles. It is readily and freely employed at the bidding of the will. Its position and relations are such as to enable it to close the outlet of the bowel, by puckering the skin, as the mouth of a bag is closed, but not so as to constitute, alone, a sufficient security against the escape of its contents, when the peristaltic movement is active.

The vertical plane of fibres, which is just within the cutaneous sphincter, is constituted of a strong annular band of considerable thickness, and of darker hue than the muscular coat of the intestine. This band is not abruptly defined, but mingles with the circular fibres higher up; and the gradual blending in colour of the intervening fibres, which is independent of their aggregation, suggests a mixed character in respect of their function, as associated with this change. The position and arrangement of this annular band, which is uninfluenced mechanically by distension above, is exactly such as to qualify it for the office assigned to it. Its nerve-supply, in contrast with that of the commencing portion of the rectum, is derived chiefly from the sacral branches of the spinal nerves. In texture and appearance it resembles the muscles supplied by these nerves, rather than those under cyclo-ganglionic control.

When the outlet of the bowel is examined, the cutaneous sphincter is found usually not firmly contracted unless irritated: but just within the superficial plane of muscle a rigidly con-



tracted annular band is felt, which seems to be uninfluenced by the introduction of the finger, and to be relaxed only in association with the effort or urgent need to evacuate the bowel, (bearing down). Defæcation is completed by its immediate after-contraction. Thus, diarrhœa relaxes this muscle, and calls into voluntary activity the cutaneous sphincter. In paraplegia from injured cord, although at first the bowel may empty itself unconsciously to the patient, constipation usually follows, due, as I believe, partly to the deterioration of spinal-reflex power in the lower third of the rectum, consequent on the lesion the cord has sustained, and also, doubtless, in part to the inability of the abdominal muscles to assist the expulsive effort, if the injury be sufficiently high up to paralyse them. When arguing, very many years since, in favour of the view that the lower part of the rectum derives special innervation from the spinal centre, I quoted the following experiment, which was performed incidentally when I was engaged, in a very humble way, in assisting Dr. Marshall Hall in his investigations. Having had the entire alimentary canal removed from a horse immediately after death, I remarked that the peristaltic movement, which was vigorous in the small and upper part of the large intestines, became less active in the rectum, until it was scarcely perceptible in its lower part. The contents of the bowel were readily expelled through an artificial opening higher up, by the rhythmic wave, which failed to reach the lowest part of the rectum and force its contents through its open mouth.<sup>1</sup>

During defæcation the sphincters are relaxed. This is an associated and necessary condition, which is exemplified in many analogous instances; notably in relaxation of the pyloric ring when the digested food is urged to apply for a passage through it; also in the varying condition of the rima glottidis in respiration, in dilatation of the nostrils in dyspnœa, in deglutition, &c.

In connection with this subject, I may notice the apparent anomaly of the inaction of the bladder simultaneously with unconscious evacuation of the intestine in crushed spinal cord, or in compression of the brain. The competency of the bowel to empty itself is often attributed solely to paralysis of

<sup>1</sup> *Anatomy and Physiology of the Nervous System*, 1836.

the sphincters; and this is correct so far as voluntary control by the cutaneous sphincter is concerned, but very little otherwise. There is really no anomaly if we consider the source of nerve-force supplied respectively to the bladder and intestine. The latter, except at the lowest part of the rectum, is under the control of the cyclo-ganglionic nerve-centres; and the peristaltic action is, therefore, undisturbed by injury to the brain or cord. It is true that the reflex power of the sphincters is impaired by the violence inflicted on the cord, as already noticed; and, as I have remarked, volition is suspended or annihilated; thus the resistance opposed to the evacuation is lessened. But so long as the lower part of the cord is intact the sphincters retain, to a very considerable extent, their reflex controlling power, though it is insufficient, without voluntary aid, to prevent the expulsive action of the intestine. It is well known that when the lumbar portion of the cord is destroyed,—as is readily shown in a decapitated animal,—all sphincter action ceases.

Should it be asked why, if the annular fibres constituting the internal sphincter are credited with the action ascribed to them, the same office is denied to the muscular fibres similarly related to the neck of the bladder, I believe the foregoing remarks and the general tenor of the preceding pages afford a sufficient answer. The reasons for this distinction may be thus briefly stated—1. The arrangement of the annular fibres of the bladder is not such as to suggest that they are designed for control at the time it is really required, viz., when the bladder is distended. In the rectum the circular fibres of the inner sphincter form around the outlet a more compact and thicker ring, which is undisturbed by the distension of the bowel above. 2. The character of the annular fibres around the neck of the bladder is identical with that of its general muscular coat, and both parts derive their nerve-supply from the same source. The ring around the lower extremity of the rectum is deeper in colour, and, instead of being supplied from the cyclo-ganglionic system, receives its principal innervation from the spinal centre. 3. There is very little need for active or muscular control at the neck of the bladder, the hydrostatic law alluded to sufficing to resist, passively, very great pressure. In the rectum the necessity for such muscular control is obvious; and especially

so as the nerve-force on which the peristaltic action depends is derived from the cyclo-ganglionic system, and is therefore independent of the cerebro-spinal centre.

The following are the physiological conclusions, whether novel or otherwise, which it has been my purpose to establish and illustrate :—

1. The muscular coat of the bladder acts under the government of the will, but is also subject to reflex influence.

2. The abdominal muscles take no necessary part in the expulsion of the urine.

3. In early life the action of the bladder is chiefly reflex, but is gradually rendered voluntary by education and habit.

4. The retaining power of the bladder is due (*a*) in great measure to the hydrostatic law, in accordance with which the egress of fluid from a reservoir through a small tube is determined; (*b*) to the elasticity and (?) muscularity of the urethra; (*c*) to its compression, whilst under the arch of the pubes, by the compressor urethræ muscle.

5. The annular fibres around the neck of the bladder have not a sphincter action.

6. Incontinence or retention of urine may be referred to excessive or deficient sensitiveness of bladder, ill-regulated control, atony, mechanical obstruction. When violence is inflicted on the nerve-centres,—either brain or spinal cord,—the bladder may be rendered partially or wholly incapable of expelling its contents. In lesion of the brain this incapacity is proportioned to the profundity of the coma, and due to insensibility and suspension of voluntary power. In compression of the cord the cause is the same, but operates by interruption of the afferent and efferent currents; and the reflex energy of the cord is also impaired.

7. The rectum is guarded at its outlet by two sphincter muscles, one cutaneous and chiefly voluntary, the other intestinal and spinal-reflex. In compression of the brain the former is almost or entirely disabled; in compression of the cord the power of the latter is likewise impaired.

8. Where an appeal is made, through common sensation, to the nerve-centres, it is not consistent with our physiological

knowledge to exclude volition from participating in the origination of the motor force which is evoked by that appeal.

In the preceding pages, the nature of my subject has compelled me to assume as probable some things which do not admit of demonstrative proof; and where this is the case, I have expressed myself accordingly. But if my views are such as to satisfy physiological criticism, I may venture to claim for them the further recommendation that they afford a reasonable explanation of some of the otherwise obscure pathological phenomena presented by the excretory urinary organs.

TEN CASES OF CONGENITAL CONTRACTION OF THE  
STOMACH, WITH REMARKS. By W. ROGER WILLIAMS,  
F.R.C.S., *Surgical Registrar to the Middlesex Hospital.*  
(PLATE XVII.)

*Case I.*—In the course of dissection at the Middlesex Hospital, in the Winter Session of 1879, I noticed a singular deformity of the stomach.

The body was that of a woman, aged 72, who died in the workhouse, a month previously, of cerebral disease and apoplexy. When alive she was remarkable for her voracious appetite. No further particulars could be obtained.

Viewed *in situ* the stomach consisted of two divisions (*vide* fig. 1, for which I am indebted to Mr Hensman), separated by a narrow contraction, nearer the cardiac than the pyloric orifice. Of these the cardiac one appeared much the larger; it was of pyriform shape, with the base above and the apex below, at the seat of contraction. The pyloric division was elongated, tapering at each end, and bulged in the middle. At the narrowing these divisions of the organ were approximated by a flexure of the small curvature, the cardiac pouch being received into the concave upper border of the pyloric one.

The organ also presented two other slight contractions, viz., one, in the cardiac division, on the side of the great curvature, marking the limit of the great cul-de-sac; and the other, in the pyloric division, on the side of the small curvature, marking the limit of the small cul-de-sac.

The former of these divisions had a shiny, membranous appearance, and in the vicinity of the cardia the longitudinal fibres from the oesophagus could be seen radiating over it, a very strong band passing to the small curvature; the latter was dark and fleshy looking, the transverse arrangement of its surface fibres being very obvious. Contrary to what is usual, the walls of the cardiac part of the organ were thicker than those of the pyloric.

With the viscus moderately distended, the length of the stricured part was  $1\frac{1}{2}$  inch, the maximum contraction being in the middle, which was distant  $4\frac{1}{2}$  inches from the cardiac orifice and  $5\frac{1}{2}$  from the pyloric, measured along the small curvature. Be-

tween the same points, along the great curvature, the distances were  $9\frac{1}{2}$  and 13 inches respectively. The contraction appeared to have been formed chiefly by an infolding of the great curvature. In its natural position, there was no obvious puckering or irregularity; but a smooth, shiny, even surface. To its upper border, however, the tail of the pancreas, unduly hard and enlarged to the size of a walnut, was adherent. The external circumference of its narrowest part was 2 inches, that of the pylorus 3 inches. Similar measurements round the largest parts of the cardiac and pyloric sacs, gave 9 inches and  $7\frac{1}{2}$  inches respectively. About an inch to the cardiac side of the maximum contraction, in the small curvature, a slight irregularity and well-marked thickening could be detected on manipulation, over an area rather larger than a shilling; in the corresponding position, on its pyloric side, a similar indurated area, only smaller, was also made out. Otherwise the contracted part, like the rest of the organ, presented no obvious lesion. As the body had been injected I dissected the various gastric arteries. It was then obvious that the vascular arches along the curvatures were interrupted, each division of the organ having a separate blood supply, whose main trunks arose in the vicinity of the cardiac and pyloric extremities; and as their terminal branches approached the seat of contraction, they rapidly dwindled away. The only communication between the two sets of vessels was by a few exceedingly minute twigs in the latter situation. After its removal from the body the stomach was fully inflated, but without producing the least alteration in the contraction. When in a state of moderate distension the cardiac pouch held 2 pints of water, the pyloric only 1 pint. It weighed 8 ounces; but this certainly conveys an exaggerated idea of its proper weight, for when put on the scales it was sodden with water. On laying the organ open, its mucous membrane presented throughout a perfectly normal appearance, with the exception that the rugæ of the cardiac pouch were unduly prominent. Both orifices were normal. The contraction was just large enough to admit a man's thumb.

A callous-looking, oval ulcer, about half an inch long, was discovered on the small curvature, at a distance of nearly an inch from the seat of maximum contraction, and to its cardiac side. The tail of the pancreas had been adherent opposite

this spot. At a corresponding position, on the pyloric side of the contraction, was a firm white cicatrix, as large as a pea, the site of a healed ulcer. There was no puckering or irregularity of the mucous membrane in either of those situations. Between them was a stretch of perfectly healthy tissue about 2 inches long, which included the whole length of the contracted area. Contraction had not taken place in the plane of either lesion, but in the intervening portion. These appearances seem to me to afford strong evidence that the lesions described were secondary to, and caused by, the contraction. In what other way can we account for their occurrence, in the positions most exposed to irritation, during the passage of food under such circumstances; and *not* in the plane of any contraction, as would have been the case had either of them *caused* such a condition?

There was nothing noteworthy about the condition of the other organs.

*Case II.*—For this specimen I am indebted to Professor Thane: he removed it from the body of an elderly male, in the University College dissecting room.

The stomach in question (fig. 2) was a rather small, thick and muscular one, weighing  $5\frac{1}{2}$  oz.

It presented a well-marked contraction, formed chiefly at the expense of the great curvature, nearer the cardiac than the pyloric orifice. There was a slight infolding of the small curvature at the seat of constriction.

The cardiac pouch was ill-developed, and much smaller than the pyloric, than which its walls were also thicker.

The usual slight contraction existed, limiting the small cul-de-sac.

No puckering or irregularity could be detected; in short, in other respects the organ appeared normal.

When moderately distended, the circumference of the largest part of the cardiac pouch measured 8 inches; that of the pyloric 10; the circumference at the seat of contraction was 6 inches.

Measured along the small curvature, the contraction was  $2\frac{1}{2}$  inches distant from the cardiac orifice,  $3\frac{1}{2}$  from the pyloric. When very fully inflated the contraction was not in the least effaced.

The mucous membrane in the cardiac pouch presented well-marked rugæ; there were none in the pyloric pouch, except near

the pyloric orifice. The latter had the appearance, not of a smooth, circular, patent ring, but of a narrow elongated passage, lined by longitudinally arranged rugæ, somewhat whorled, continued from the stomach into the duodenum for a short distance.

No induration, scar, ulcer, or any other lesion could be detected in any part of the organ.

*Case III.*—Mr. Shattock kindly called my attention to this specimen, No. 5513 in the University College Museum, being the stomach of a woman who died of cirrhosis of the liver.

The organ (fig. 3), which was small and elongated, consisted of a cardiac and a pyloric division, separated by a well-marked contraction, distant  $2\frac{1}{2}$  inches from the cardiac, and  $6\frac{1}{2}$  from the pyloric orifice, measured along the small curvature. The cardiac division was saccular in shape; the pyloric intestiniform—the latter being the larger and thicker. In other respects, as viewed from the outside, the stomach appeared normal. There was no sign of any adhesion between it and adjacent structures.

Internally, the mucous membrane in both divisions presented very prominent longitudinal rugæ. The cardiac and pyloric orifices were normal.

Situated in the small curvature, about an inch apart, were two small scars, one on either side of the constriction, from which each was equally distant.

Each scar was the size of a threepenny piece—smooth and whitish. The intervening tissues were perfectly normal; no puckering or irregularity could be detected. The organ presented no other lesions. The contraction was large enough just to admit the thumb and index finger.

*Case IV.*—No. 1795 in Guy's Hospital Museum. In the catalogue is the following brief description:—"Stomach showing hour-glass contraction about its centre." This specimen and the succeeding one are very old; they were, I believe, presented to the museum by Sir Astley Cooper. Dr. Wilks alludes to them in his work on pathological anatomy. Neither specimen had been previously laid open and thoroughly examined. By the kind permission of Dr. Fagge, I was able to do this some time ago, and in the presence of Dr. Carrington, who has since exhibited them at the Pathological Society of London, and included them in his paper in the *Transactions*, forgetting, I am sur-



prised to find, to mention this circumstance—accidentally, no doubt.

The organ I am about to describe (fig. 4) was a little under the usual size; externally it presented no obvious morbid appearance, other than the contraction, which was smooth, even-looking, and about an inch long. Of the two pouches, the cardiac was the larger. The walls of both were thin, and uniform in this respect throughout.

The centre of the contraction was distant  $2\frac{1}{2}$  inches from the cardiac orifice, and 4 inches from the pyloric, measured along the small curvature.

In the latter part, two thin, flat, hard, oval plates, feeling almost calcareous, were detected,—one on either side of the contraction, separated by an intervening portion of perfectly healthy tissue, rather more than an inch in length.

The interior of the stomach had a normal appearance. No trace of any lesion could be seen. The plates in the small curvature were covered by healthy mucous membrane, beneath which they were situated. They were the result probably of chronic inflammation, excited by the undue impact to the food in these situations, owing to the existing deformity. The fact that they occurred in the precise localities so frequently occupied by ulcers in such cases, is an additional reason for believing that the latter usually originate, under similar circumstances, in this manner.

The contraction was very small, barely admitting the tip of the little finger. The cardiac and pyloric orifices were normal.

*Case V.*—No. 1796 in Guy's Hospital Museum. In the catalogue it is described as—"Stomach showing hour-glass contraction about its centre; peritoneal surface at that part thickened and white."

The organ (fig. 5) was rather above the normal size; and the cardiac division was larger than the pyloric. Its walls were thick, but there was no appreciable difference between the two divisions in this respect.

The contraction was of the annular variety, and externally it presented a smooth and regular appearance.

An inch to its left, close to the small curvature, was a small stump of fibrous tissue, as if an adhesion had once existed here between it and some adjacent structure. At this spot there was

an indurated area about the size of a sixpence. In the corresponding position, on the pyloric side of the contraction, a similar indurated patch could be felt. In this case, as in the previous ones, no lesion could be detected at the seat of maximum contraction. The distance from the latter to the cardiac orifice was 4 inches, to the pyloric  $4\frac{1}{2}$ , measured in each case along the small curvature. On examining the interior of the organ a smooth, whitish patch was discovered, opposite each indurated area—to which the mucous membrane, elsewhere freely movable over the subjacent structures, was adherent; and around these spots were some radiating rugæ, suggesting the appearance of old scars. No trace of any other lesion. The stricture in this case only just admitted the end of an ordinary lead pencil. The cardiac and pyloric orifices were normal.

*Case VI.*—Dr. Carrington found this specimen in the Guy's Hospital dissecting-room. He exhibited it at the Pathological Society last winter. I am indebted to him for allowing me to examine it.

This stomach (fig. 6) was rather small and thin-walled—the cardiac pouch being rather the more capacious.

The centre of the contraction was distant  $2\frac{1}{2}$  inches from the cardiac orifice, and  $4\frac{1}{2}$  from the pyloric, measured along the small curvature.

There was a slight patch of thickening in the latter position about three quarters of an inch to the cardiac side of the contraction.

On examining its interior no definite scar could be detected at this spot; but here the mucous membrane was smooth and not so freely movable on the subjacent parts as elsewhere. No other abnormal appearance, either externally or internally, could be made out. The stricture just admitted the thumb. Cardiac and pyloric orifices normal.

Dr. Carrington has suggested that some of the thickenings in the last cases may be due to the injected gastric arteries; but, independently of the fact that in at least two of the specimens the arteries had not been injected, the facts do not warrant such a supposition, for, as I have previously mentioned, in these cases of congenital contraction the arteries in this position are either non-existent or dwindled to the tiniest twigs.

*Case VII.*—No. 11·6B of the Physiological Series of St. Bartholomew's Hospital Museum.

An inflated, dried stomach (fig. 7), of small size, divided into a cardiac and pyloric portion by a well-marked constriction, situated at a point  $3\frac{1}{4}$  inches from the cardiac orifice, and  $5\frac{1}{4}$  from the pyloric, measured along the small curvature. I judged the contraction large enough to admit the thumb and little finger. The pyloric division was much elongated, and as it approached the valve resembled a piece of small intestine. Its walls were of about normal thickness. On each side of the contraction, in the small curvature, about an inch apart, was a small darkish patch, of less size than a pea, probably the seat of some former lesion. The pyloric valve was very ill developed—forming a scarcely perceptible ring.

In the specimen next preceding this in the Museum (No. 11·5) I regard the contraction as pathological.

I am indebted to Mr. Bowlby for kindly allowing me to examine these specimens, and the succeeding one.

*Case VIII.*—A. 216 of the Teratological Series of St. Bartholomew's Hospital Museum. This is the case recorded by Mr. Morrant Baker in the *Transactions of the Pathological Society of London*, vol. xvii. p. 105. A dry stuffed specimen (fig. 8) the arteries of which had been injected: the separate arterial supply of each sac, and the other peculiarities I have mentioned, were nearly as obvious as in Case I.

In the catalogue it is described as—"A stomach, a narrow portion of which, nearly midway between the cardiac and pyloric orifices, is extremely contracted, so as to divide the organ into two compartments, which communicate only by a small orifice. The stomach appears otherwise healthy. From a woman, aged 63, in the dissecting room."

The organ was of rather large size—the pyloric pouch exceeding the cardiac in this respect. The walls were of about normal thickness throughout.

The contraction was of the annular variety, and presented no thickening, irregularity, or other sign of disease. It was distant 4 inches from the cardiac extremity and 7 inches from the pyloric, measured along the small curvature. The index finger could just pass through it.

No lesion could be detected in any part of the organ.

*Case IX.*—A wet specimen, No. 207 in St. George's Hospital Museum, and presented by Dr. Page.

A medium-sized stomach (fig. 9), having a well-marked contraction, about 1 inch long, the centre of which was distant from the cardiac orifice 3 inches and from the pyloric  $4\frac{1}{2}$ , measured along the small curvature. The pyloric division was considerably larger than the cardiac. In the neighbourhood of the contracted part the fibrous membrane was slightly opaque and thickened. Externally there was no other obvious lesion.

Each division had been opened, so that a good view of the interior could be obtained. On the cardiac side of the contraction, near the small curvature, at a distance of 1 inch and  $1\frac{1}{2}$  inch respectively from it, were two small superficial erosions. Otherwise the interior of the organ presented a perfectly normal appearance. There was no lesion in the pyloric pouch. The stricture admitted the little finger, and was itself quite free from disease.

In the catalogue it stated that—"when recent the membrane was very vascular and in places blood was extravasated beneath it. The other organs were healthy. The specimen was removed from the body of a woman who for many years had suffered from nausea, vomiting after taking food, cough in winter, and dyspnoea. Two or three days before death she complained of tenderness at the epigastrium on pressure; and for forty-five hours previously she was delirious."

*Case X.*—There is a wax model of this specimen, by Professor Chaussier, in the Musée Dupuytren at Paris. The stomach, a rather large one, presented a marked contraction in the great curvature, at the junction of the great cul-de-sac with the body of the organ. On its outer surface, at the top of the great cul-de-sac, was a small abrasion, where it had once probably been adherent to the under surface of the diaphragm. There was no other lesion.

I believe this list includes all the specimens of the kind contained in the Metropolitan Museums.<sup>1</sup>

<sup>1</sup> Since this was written I have discovered another specimen in the Museum of the London Hospital. This specimen is particularly interesting, as, in addition to the well-marked hour-glass contraction, the stomach also presents contractions separating the great and small *cul-de-sacs* from the rest of the organ.

Dr. Charlewood Turner, who has examined the specimen, has kindly informed me that it presents no trace of any ulcer, scar, or other lesion.

## REMARKS ON CONGENITAL CONTRACTION OF THE STOMACH.

The old anatomists were aware of the occasional occurrence of contractions at about the middle of the stomach, and they attributed them for the most part to pathological causes.

Judging, however, from the confused and mixed-up descriptions of this peculiar deformity which appeared at that time, their knowledge of the subject was very vague and indefinite. The first well-recorded cases with which I am acquainted are to be found in Morgagni's stupendous work, *De Sedibus et Causis Morborum per Anatomen Indagatis*:<sup>1</sup> reference is there made to cases previously described by Blasius, Valsalva, Riolanus, and other authors of that period.

Monro<sup>2</sup> notices Morgagni's cases, and gives the chapter and verse of others by Lorry, Walter, Ludwig, Hufeland, and Van der Kolk.

Sir E. Home's<sup>3</sup> observations, on the shape and structure of the stomach in various animals and in man, threw additional light on the subject; but he failed to recognise the congenital nature of many of these contractions.

This was reserved for Professor Struthers,<sup>4</sup> who in a very interesting paper has recorded two cases of this nature. I am indebted to him for many valuable suggestions. These cases, together with those I have described, are the only ones of the kind of which I have been able to find anything like a complete record. Having visited the various metropolitan museums, I have discovered in them only six such specimens: the Hunterian Museum not containing a single example. Hence we may conclude that the affection is one of exceeding rarity; for it is hardly probable that a striking deformity of this kind, in such an organ as the stomach, would escape unnoticed, even at the most casual *post-mortem* examination.

It is remarkable, however, that hitherto most of these specimens have come from the dissecting room. On studying their history, we are struck with the frequency with which the contracted stomach is the seat of lesion,—either ulcer or cicatrix. It was this no doubt that misled the older authors. May be

<sup>1</sup> *Vide Eng. Trans.*, Letter xvi. art. 38; xxvi. art. 31; xxx. art. 7; xxxvi. art. 3.

<sup>2</sup> *Lectures on Human Anatomy*, by Alex. Monro, jun., 1813, vol. ii. p. 111.

<sup>3</sup> *Lect. on Comp. Anat.*, also *Phil. Trans. Roy. Soc. Lond.*, 1807 and 1817.

<sup>4</sup> *Anat. and Physiol. Obs.*, art. on "Double Stomach."

there are some who still think as they did, that in such cases these contractions have a pathological origin.

A careful examination of the subject will, I think, lead to a different conclusion. It would be contrary to my own experience, and to that of the best observers, to deny the existence of stomach contractions due to ulceration and its results. The museum of St. Thomas's Hospital is particularly rich in specimens illustrative of this fact. The question is how to distinguish one kind of contraction from the other. I believe this may be readily done by paying attention to the following facts:—The congenital contractions, however long and narrow they may be, and they are generally longer and narrower than those of the other form, present both externally and internally a perfectly smooth and normal appearance; whereas the pathological ones are irregular and puckered, with thickened cord-like rugosities radiating from the seat of disease.

Again, the situation in which these contractions are so uniformly found, viz., nearer the cardiac than the pyloric extremity, is not that generally affected by gastric ulcers, which, as Rokitsky<sup>1</sup> has remarked, are usually seated in the middle zone of the pyloric half of the stomach. It should be remembered also, that although ulceration occasionally leads to contraction, such an occurrence is quite exceptional.

When, however, this result does ensue, the contraction is always situated in the plane of the lesion. In this respect then, such contractions differ markedly from those of a congenital nature associated with ulceration; for in the latter no contraction is to be found in the plane of the lesion, and no lesion in the plane of the contraction.

Thus, in every one of my cases I found the whole length of the contracted area perfectly free from any sign of disease. Such lesions as existed were situated in the neighbourhood of the small curvature, either on the cardiac or pyloric side of the contraction, not unfrequently in both places; in the latter circumstance the intervening contracted portion, an inch or an inch and a half in length, was perfectly healthy in every case.

I submit that these appearances only admit of one explana-

<sup>1</sup> *Manual of Pathological Anatomy*, Syd. Soc. Transl., vol. ii. pp. 23 and 24.

tion, namely, that the lesions were really caused by, and were secondary to, the contractions.

Against their congenital origin, it may be urged that no examples of this deformity have as yet been discovered in young children, at least I am not aware of any; nor is this surprising, considering the rarity of the affection and the infrequency of *post-mortem* examinations at that age. Morgagni<sup>1</sup> has, however, recorded a remarkable case in which symptoms of this affection appeared at birth, and were hereditary in three successive generations. Hence I think too much importance should not be attached to such a purely negative objection.

The greater relative frequency of these contractions in women, and their apparent absence in infants, led Scemmering to attribute them to pressure of the stays. But if this were the case they would be of common occurrence in that sex, which is not the case; besides, they are met with also in men.

Other lesions besides those resulting from ulceration may, no doubt, in very exceptional circumstances, give rise to hour-glass contraction of the stomach. No. 115 of the Physiological Series of the Museum of St. Bartholomew's Hospital is a case in point. It is classed with a specimen of congenital contraction, which it much resembles, and is described in the catalogue as an example of the form which the stomach assumes during the later period of digestion. Mr. Bowlby, the curator, kindly allowed me to make a thorough examination of it. At the middle of the organ was a smooth-looking contraction,  $2\frac{1}{2}$  inches long, and about the size of a man's thumb. The contracted part felt perfectly solid; at this place its walls were greatly hypertrophied. Internally no lesion could be detected; but the mucous membrane at the contracted part presented numerous longitudinal lamelli-form rugæ closely pressed together. Microscopical examination of a section of the thickened wall revealed what I believe to be an early stage of cancerous infiltration, but in this particular Mr. Bowlby does not agree with me.

Having thus established the congenital nature of these contractions, it may be interesting to inquire whether they have any counterpart in the normal condition of the stomach.

Anatomists have generally overlooked the occurrence of

<sup>1</sup> *Op. cit.*, Exp. xxx. art. 7.

slight contractions at certain definite situations in the normal stomach, although these may be frequently seen.

Home<sup>1</sup> regarded the human stomach, like that of many animals, as naturally the seat of such a contraction at about its middle; though he failed to detect any increased development of the muscular fibres at this spot.

Most anatomists admit a slight contraction a little to the left of the pylorus, and one has occasionally been observed separating the great cul-de-sac from the body of the stomach.

On this subject Cruveilhier<sup>2</sup> remarks:—"At about an inch from the pylorus the stomach is markedly flexed on itself, forming a very pronounced bend, *coude de l'estomac*, and presenting a pouch on the side of the great curvature, with a corresponding internal excavation, described by Willis as the *antrum pylori*, and by others as the small cul-de-sac or small tuberosity of the stomach. It is not rare to see a second pouch beside the first, and a third smaller one, on the side of the small curvature, owing to the bend which the stomach takes. These pouches, hardly preceptible in many subjects before insufflation, are rendered very distinct, and even in some cases very considerable, by distension."

Henle, in his treatise on Anatomy, says:—"The *antrum pylori* is separated from the rest of the stomach by a contraction near the pylorus, more marked on the upper than on the under surface. A second contraction is rarely found at the middle of the stomach, which divides it into a right and left half."

Such are the opinions of these authors, from which, together with my own observations, I conclude that the human stomach is naturally divided by three contractions into four compartments, viz., one at each extremity, the small cul-de-sac and the great cul-de-sac; and an intermediate portion, subdivided by a contraction at about its middle, into a cardiac and pyloric portion, this last contraction being, as a rule, less obvious than the others.

Careful examination of a considerable number of congenitally contracted stomachs has revealed the interesting fact, that these contractions do not occur just anywhere, but only in certain

<sup>1</sup> *Phil. Trans. Roy. Soc. Lond.*, 1807, p. 170.

<sup>2</sup> *Anatomic Descriptive*, tome ii., ed. 1834, p. 463.



definite situations, which are identical with those occupied by the normal contractions.

As my cases testify, the commonest seat of such abnormal contractions is at about the middle of the organ. In two of Morgagni's <sup>1</sup> four cases the contraction was in this situation : in the other two it occupied such a position as to separate the antrum pylori from the rest of the stomach.

Rokitansky <sup>2</sup> remarks :—" Among the abnormalities of the human stomach, we reckon first all those rare congenital malformations, in which an annular contraction divides the organ into a cardiac and pyloric portion, or in which two or three such contractions form three or four sacculated divisions, and thus cause in these a resemblance to the stomach of ruminants. We distinguish from the contractions produced during the agony of death, by the fact that the latter may be removed by inflating the stomach."

Brinton <sup>3</sup> says :—" These transverse subdivisions of the tube may possibly imply a mere arrest of development in the site of imperfect septa."

In Luton's <sup>4</sup> opinion this anomaly resembles the multiple stomachs of ruminants, especially of the cetaceous herbivora. With these ideas he cites the example recorded by Billard of a stomach whose mucous membrane resembled in appearance that of the reticulum of a ruminant, and bristled with long villousities.

In one of my cases the contraction was so situated as to divide the great cul-de-sac from the rest of the stomach.

Cruveilhier, <sup>5</sup> Handfield Jones, <sup>6</sup> Leube, <sup>7</sup> and other authors allude to these contractions in similar terms.

It is interesting to notice the occurrence of various temporary contractions in precisely the same localities as these congenital ones ; and, like them, commonest at about the middle of the organ. Home <sup>8</sup> thought the human stomach naturally presented

<sup>1</sup> *Op. cit.*

<sup>2</sup> *Op. cit.*, vol. ii. p. 30.

<sup>3</sup> Todd's *Encyclopædia of Anat. and Physiol.*, art. "Stomach," &c., in the Supplement.

<sup>4</sup> *Nouveau dict. de med. et de Chir. prat.*, art. "Estomac."

<sup>5</sup> *Op. cit.*, tome ii. ed. 1834, pp. 457-8.

<sup>6</sup> *Manual of Path. Anat.*, p. 494.

<sup>7</sup> Ziemssen's *Encyclopædia*, art. "Stomach."

<sup>8</sup> *Lect. on Comp. Anat.*, vol. ii. fig. xii. and context ; also *Phil. Trans. Roy. Soc. Lond.*, 1807, pp. 157, 170 and 171, 1807-1817, p. 350.

a temporary contraction of this kind during the process of digestion ;] he pointed out that the bilocular form of stomach is not unfrequently met with in bodies examined immediately after death, and in such as have met with sudden death during the process of digestion.

Dr. Struthers<sup>1</sup> fixed the usual locality of this contraction at 4 or 5 inches to the left of the pylorus.

A remarkable case of temporary contraction was recorded some years ago by M. Broca :<sup>2</sup> the specimen was obtained from the body of a female criminal who had been executed. The great cul-de-sac presented a spherical dilatation, succeeded by a contraction. Setting out from this point the stomach was uniformly narrowed, but another contraction occurred before the pylorus was reached, so that the organ resembled somewhat a portion of large intestine. Here there was an appearance of three stomachs. "This specimen," said M. Broca, "is also of interest, because it has much analogy to another which I met with in a male criminal who was executed, and of which I am now able to show you the drawing. With regard to contraction of the stomach, internal to the great cul-de-sac, we can easily conceive how it might be produced. The longitudinal fibres of the stomach, prolonged below the great cul-de-sac, form a loop, which, in contracting, would produce such a result." The literature of the subject reveals several other cases of a similar description. In accordance with the above facts, I think we may regard the occurrence of temporary and permanent contractions, dividing the stomach into two, three, or four portions, as due to exaggeration of the slight contractions normally existing in these situations.

Whether the human gastric mucous membrane presents structural differences, corresponding to these subdivisions, is at present an open question.

That of the dog and some other animals has been found to possess in the cardiac region only peptic glands, and in the pyloric only mucous ones. In these animals Ebstein has described a so-called intermediary zone, in which the two kinds of glands are not only mixed, but present also important structural modifications. Heidenbain has shown, by isolating

<sup>1</sup> *Op. cit.*

<sup>2</sup> *Bulletin de la Soc. Anat.*, t. xxvi., 1851, p. 30.

various segments of the organ, that the fluid secreted by one part differs from that secreted by another.

With regard to the human stomach, however, most observers have not ventured beyond the vague foreshadowing of somewhat similar distinctions.

Cruveilhier,<sup>1</sup> however, with his usual perspicacity, has pointed out several differences obvious to the naked eye. Thus he says:—"There is one fact to which too much importance cannot be attached, and that is the difference in the appearance of the mucous membrane of the great tuberosity of the stomach, and of the part situated to the right of the œsophagus. Sometimes the line of demarcation forms a perfect circle; and this is very remarkable, because in man, who has a single stomach, it may be regarded as a rudiment of the division into the compound stomachs found in the lower animals: for a multiple stomach results rather from some difference in the structure of the mucous membrane than from the existence of different compartments or distinct cavities."

In another place<sup>2</sup> he describes the mucous membrane of the pyloric region, as differing from that of the cardiac, in presenting frequently a granular aspect, similar to that seen in the stomach of the pig.

Disease, he says<sup>3</sup> often respects the line of demarcation existing between these two portions. In confirmation of this statement Dr. Philip<sup>4</sup> has recorded the case of a woman who ate and digested her food perfectly to the last; but whose stomach was found, after death, to be ulcerated everywhere, except at the cardiac end.

Taken in conjunction with the remarkable proneness of the cardiac pouch to *post-mortem* digestion, as described by John Hunter, the above fact tends to confirm Home's opinion that this region is the exclusive seat of the peptic glands.

The mucous membrane here is thinner, softer, and more vascular than in other parts of the stomach, and it is more firmly adherent to the subjacent structures, so that it can only

<sup>1</sup> *Op. cit.*, t. ii., pp. 478-4-8; *vide* also his remarks with regard to bilocular stomachs, *ibid.*, pp. 467-8.

<sup>2</sup> *Op. cit.*, t. ii. p. 478.

<sup>3</sup> *Op. cit.*, t. ii. p. 475.

<sup>4</sup> *An Experimental Enquiry into the Laws of Vital Functions*, ed. 1817.

be separated with difficulty, and it generally comes off in shreds ; whereas that lining the rest of the organ is loosely attached, and can easily be separated entire.

The development attained by this part of the stomach is subject to many variations.

Cruveilhier says :<sup>1</sup>—" It is almost entirely absent in the carnivora, very large in the herbivora, and of medium size in man. Its size varies much in different individuals. I have seen human stomachs in which the great cul-de-sac did not exceed in size that of the carnivora."

Meckel relates having seen this part of the organ absent, and replaced, as it were, by an exaggerated development of the small cul-de-sac.

Todd and Bowman remark :<sup>2</sup>—" We have suspected that the digestive power of the two parts must differ, that the office of the pyloric tube resembles that of the stomach cells generally and is different from that of the true stomach tube, that perhaps the acid product of the stomach may be furnished by one rather than the other. We confess, however, that we have been unable, on the one hand, to obtain stomachs sufficiently fresh and healthy to test the fact of the anatomical distinctness of the two regions in man, or, on the other, to ascertain the value of the conjectures just alluded to, as applied to animals in which the twofold structure is certain."

No one has expressed this view more forcibly and with greater clearness than Dr. Draper.<sup>3</sup> He says, after reviewing the structure of the stomach in different animals :—" So far from there being anything in contradiction to the doctrine that different portions of the digestive surface of the mucous membrane of the stomach are devoted to different duties, there is strong evidence in support of its truth, derived partly from the instances furnished by comparative anatomy, and partly from the anatomical structure of the gastric mucous membrane. The four separate digesting chambers of the herbivora are merely an elaboration of the structure which is presented by the apparently homogenous mucous surface in man. But that this mucous surface is in reality heterogenous, and in different regions possesses different

<sup>1</sup> *Op. cit.*, t. ii. p. 462.

<sup>2</sup> *Physiological Anatomy*, ed. 1856, vol. ii. p. 195.

<sup>3</sup> *Physiology*, 2nd ed. 1861, pp. 60-61.

powers, is shown by the fact that at one part it presents mucous follicles, at another peptic follicles, at another follicles for secretion of hydrochloric acid. As we approach towards the pylorus, the existence of a new function is betrayed by the appearance of a new mechanism—the villi—which have been so well studied by Dr. Neill; and this is even indicated externally, as shown in the accompanying diagram, where the lesser tuberosity of the stomach is separated from the rest of the organ by a well-marked constriction, showing, according to Professor Retzius, that the antrum pylori of the older anatomists is really a special compartment of the general cavity. This part of the stomach is distinguished by the greater thickness of its muscular coat, the more copious glandular development, and the presence of the well-known *plicæ fimbriatæ*.

The commencement of the duodenum also forms a special rounded cavity, which Professor Retzius proposes to name *antrum duodeni*, characterised internally by the absence of *valvulæ conniventes* and the dense array of Brunner's glands beneath its mucous membrane. This part constitutes what has been called the fourth stomach of the porpoise and of some other cetaceans."<sup>1</sup>

As to whether any such structural differences existed in my specimens of contracted stomach I am unable to state, since they were too old for such particulars to be satisfactorily ascertained either with the naked eye or the microscope. From such examinations as circumstances permitted nothing of the kind was observed.

Notwithstanding what has been said of the analogy of the human type of stomach to that of ruminants, rodents, and other herbivora, there is one important difference between them, to which sufficient attention has not been paid.

I allude to the direct passage of the food in the former, from the gullet, into the true digestive, glandular, or cardiac division of the stomach, and its subsequent escape into the mucous, muscular, or pyloric division: an arrangement just the converse

<sup>1</sup> In this connection such cases as that recorded by Dr. Goodhart a few years ago in the *Transactions of the Pathological Society*, in which the upper part of the duodenum was almost completely separated from the rest by a congenital contraction, are of interest. I have since seen a fetus in which these parts of the duodenum were completely separated—each ending in a *cul-de-sac*.

of that observed in the latter. In this particular, the human type of stomach appears to resemble somewhat that of birds rather than ruminants; unless indeed the crop of birds is regarded as analogous, not to the cardiac portion of the human organ, but to the cesophageal dilatations which form the chief part of the ruminant stomach—a view which, I believe, is not generally entertained, though it seems to me not altogether improbable.

Lastly, it remains to inquire what relation, if any, these contractions bear to the alterations in shape which the stomach undergoes during the process of natural digestion. In the bodies of human beings and of various animals, examined very soon after death, especially after sudden death during the process of digestion, as previously mentioned, Home frequently found hour-glass contractions of the stomach; but he was unable to detect, in the human stomach, any increased development of the muscular fibres in this situation, similar to what he had observed in dogs, &c. He concluded that this contraction resulted from the natural action of the organ during digestion, and that it served to maintain the more solid and undigested portions of food in the cardiac pouch, allowing only the more fluid and digested portions to escape into the pyloric division, thence to be propelled into the duodenum.

The existence of this contraction has been denied by some authors. Müller<sup>1</sup> says:—"The central constriction, of which the stomach was by Sir E. Home imagined to be the seat during digestion, has never been really observed. Neither Tiedemann nor I have seen anything of the kind in dogs." The concurrent testimony of subsequent observers is, however, against the accuracy of this statement. Brinton<sup>2</sup> is very explicit on this point. In his complete and graphic account of the movements of the stomach during digestion, as he observed them in the dog, he distinctly states that he witnessed this hour-glass contraction. Like Home, he failed to detect in the human stomach any increased development of the muscular fibres at this spot. Referring to the so-called transverse band described by Spallanzani and Haller, he says:—"Anatomy fails to recognise a

<sup>1</sup> *Vide* his *Physiology*, English trans., vol. i. p. 504.

<sup>2</sup> Lectures in *Med. Gazette*, 1849, Lect. on "Diseases of Stomach," ed. 1864, pp. 10-12; also article "Stomach," &c., Todd's *Encyclopædia of Anat. and Physiol.*, in the Supplement.

distinct band or even any exaggeration of the ordinary circular fibres sufficient to merit separate description."

Beaumont's<sup>1</sup> observations on the celebrated Alexis St. Martin, clearly prove the existence during digestion of such a contraction in the human stomach. He says:—"In attempting to pass a long glass thermometer tube through the aperture into the pyloric portion of the stomach, during the later stages of digestion, a forcible contraction is first experienced at this point (*i.e.*, 3 or 4 inches from the smaller end), and the bulb is stopped. In a short time there is a gentle relaxation, when the bulb passes without difficulty, and appears to be drawn, quite forcibly, for 3 or 4 inches, towards the pyloric end. This ceases as soon as the relaxation occurs, and the tube rises again of its own accord 3 or 4 inches, when the bulb seems to be obstructed from rising further; but if pulled up an inch or two, it moves freely in all directions in the cardiac portion."

All observers agree in describing the movements of the stomach during digestion as very different in its cardiac and pyloric portions; and this difference coincides with the varied distribution of the muscular fibres. Thus in the cardiac division, where they are only slightly developed, the movements are scarcely perceptible, whilst in the pyloric division, where they are very abundant, forcible movements occur.

It is evident therefore that during digestion the human stomach is the seat of a contraction at about its middle, which corresponds in position with the temporary and permanent hour-glass contractions of the organ to which we have previously alluded.

Whether similar contractions occur in other situations, during the natural action of the organ, corresponding to those occasionally met with after death, is at present uncertain. I have found no mention made of such contractions by those who have investigated the subject; but from the curious forms of temporary contraction with which we are familiar, I am disposed to regard their occurrence as extremely probable.

#### EXPLANATION OF PLATE XVII.

Fig. 1. *z*, seat of ulcer; seat of erosion.

Figs. 3, 5, 6, 7. The dots are seats of scars.

Fig. 4. The rows of dots are seats of hard plate.

Fig. *xx*, small erosions.

<sup>1</sup> *Experiments and Observations on the Gastric Juice*, ed. 1838, pp. 101-105.

**A NEW RULE OF EPIPHYSES OF LONG BONES.** By  
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IN studying human osteology, few points strike one more forcibly than the apparently disorderly manner in which the centres for the epiphyses of long bones appear. Two are visible at birth—one for the condyles of the femur, the other for the upper extremity of the tibia. The remaining centres appear at various intervals up to the fourteenth year, at which date the lesser trochanter usually commences to ossify.

Although anatomical history shows the subject of epiphyses to have been studied from very early times—Galen even marked the distinction between “epiphyses and apophyses”—scarcely anything has been accomplished to show why one terminal cartilage should possess an osseous nucleus earlier than another, even though they are situated on the same bone, or on the same extremity of the bone. The femur affords an excellent example of this, it being possessed of four secondary centres, three of which are situated at the proximal and one at the distal end. The ossific nucleus for the condyles is visible at the commencement of the ninth month of intra-uterine life, but of the three for the upper extremity of the bone that for the head appears in the first year after birth, that for the great trochanter is delayed until the fourth year, whilst the lesser trochanter shows no trace of ossific matter before the thirteenth or fourteenth year.

It is interesting to note the regularity with which the more important epiphysial centres make their appearance, particularly in those terminal cartilages which ossify, each from a single nucleus, and bear a large ratio to their shafts.

With the hope of finding some explanation why certain secondary centres should appear before others, I commenced a series of experiments to determine if any relation existed between the terminal cartilage and the diaphysis that would in any way offer a satisfactory elucidation of the facts above mentioned. Careful examination of numerous skeletons of fetuses at birth induced me to state the following general rule:—



*The centres of ossification appear earliest for those epiphyses which bear the largest relative proportion to the shafts of the bones to which they belong.*

The rule applies to the following bones more particularly :—

*Upper Limb.*—Humerus, radius, ulna, metacarpals, and first row of phalanges.

*Lower Limb.*—Femur, tibia, fibula, metatarsals, and first phalanx of the hallux.

It must be obvious that the remaining phalanges of hand and foot are too small at birth to admit of the application of the rule with certainty.

*Method of applying the Rule.*—The long bones and their cartilages are carefully cleaned of periosteum, perichondium, and connective tissue.

- (1) The entire bone is weighed.
- (2) The weight of each terminal cartilage is then ascertained separately.
- (3) Lastly, the shaft of the bone is weighed.

These weights known, one is able to calculate readily the proportion any given terminal cartilage bears to the shaft.

The tibia affords a good example :—

The entire bone weighed	.	.	.	147 grains.
Upper cartilage	„	.	.	48 „
Lower cartilage	„	.	.	18 „
The diaphysis	„	.	.	81 „

∴ Upper cartilage, 48 grains : shaft 81 grains :: 1 to 1·7 (nearly)

∴ Lower „ 18 „ : „ 81 „ :: 1 to 4·5.

The centre for the upper terminal cartilage may be seen as a small earthy spot at *birth*; that for the lower end is visible about the end of the second year. It is usually stated that the centre for the head of the tibia is not constantly found at birth, but in all cases where I have been sure that the fœtus was born at full term, the nucleus has been present.

Observations were first made on certain femora and tibiæ taken

indiscriminately. Finding the result in each case uniform, experiments were made upon seven skeletons, under similar conditions. In each particular instance the terminal cartilages bore the same relative proportion to their respective shafts, whatever the weight of the foetus, care being taken to obtain variety in this respect—the largest foetus weighed 12 lbs., the smallest 7 lbs.

The observations were repeated on three other skeletons, in addition to numerous single bones taken at random, with similar results.

In the instance of the femur at birth, the various cartilages attached to it weigh collectively almost as much as the diaphysis, *e.g.*:—

An average femur at birth weighs . . .	255 grains
The cartilages . . . . .	126 „
The shaft . . . . .	130 „

leaving a difference merely of 4 grains. In two instances the cartilage weighed more than the osseous shaft.

No other long bone possesses this preponderance of cartilage.

The following table contains the relative weight of bone and cartilage for the more important long bones of an ordinary skeleton at birth:—

Name of Bone.	Weight of Bone Entire.	Weight of Shaft.	Weight of Cartilage.
	grains.	grains.	grains.
Lower Limb—			
Femur, . . . . .	255	130	126
Tibia, . . . . .	147	81	66
Fibula, . . . . .	29	20	9
Metatarsal of hallux, <sup>1</sup> . . . . .	8	6	2
First phalanx of hallux, <sup>1</sup> . . . . .	3	2½	½
Upper Limb—			
Humerus, . . . . .	130	71	59
Radius, . . . . .	34	20	14
Ulna, . . . . .	35	25	10
Metacarpals, <sup>1</sup> . . . . .	6½	5	1½
First phalanx of pollex, <sup>1</sup> . . . . .	3	2½	½

<sup>1</sup> In the case of the metatarsal bone and first phalanx of the hallux the proximal cartilages only were compared; the distal cartilages were weighed with the shafts. The metacarpal and first phalanx of the pollex were examined in the same manner.

In the remaining metacarpals the distal cartilages were taken, leaving the proximal cartilage to weigh with the shaft.

The ratios borne by the separate cartilages to their respective shafts are arranged in a tabular form, on reference to which it will be readily observed that the proportion of any given cartilage to its shaft, and the dates of the development of the epiphysis, are in uniform relation.

TABLE OF PROPORTIONS AND DATES.<sup>1</sup>

Age at which Ossification for Epiphysis commences.	Name of Epiphysis.	Ratio to Shaft.
9th week of intra-uterine life,	Lower end of femur, .	1 to 1·6
At birth, . . . . .	Upper end of tibia, .	1 to 1·7
End of 1st and commencement of 2nd year, . . . . .	Lower end of radius, .	1 to 2·2
	Head of femur, .	1 to 3
	Head of humerus, .	1 to 3
	Lower end of fibula, .	1 to 3
End of 2nd year, . . . . .	1st metatarsal, .	1 to 3
	Lower end of humerus, .	1 to 3·4
3rd year, . . . . .	1st metacarpal, .	1 to 3·5
	1st phalanx of hallux, .	1 to 4
	Lower end of tibia, .	1 to 4
End of 3rd year, . . . . .	Head of fibula, .	1 to 4·6
4th year, . . . . .	Great trochanter, .	1 to 6
5th year, . . . . .	Lower end of ulna, .	1 to 6
5th to 6th year, . . . . .	Upper end of radius, .	1 to 6·5
14th year, . . . . .	Lesser trochanter, .	1 in 30

The humerus is probably the least accurate in the list, on account of the difficulty in separating the various pieces of cartilages of the great tuberosity and the head. The inferior extremity could not be divided, consequently the ratio of the entire lower epiphysial cartilage is placed opposite the date of appearance of its earliest nucleus (that for the capitellum), which becomes developed about the third year.

At birth the upper end of the ulna consists of a large mass of cartilage, its ratio is not given in the list, because growth from the shaft invades this terminal cartilage until nothing but a thin lamella remains for independent ossification at the tenth year.

The intention of this paper is to show that the ratio of a terminal cartilage to the diaphysis being given, on referring to the

<sup>1</sup> The dates are taken chiefly from 9th edition of Quain's *Anatomy*.

table the date of appearance of its ossific centre may be inferred with a tolerable amount of accuracy. In certain of the bones the ratio varies, sometimes being a little in excess, sometimes a little below the figures given in the tables, but rarely more than a few points above or below the theoretical quantity. This occurs most frequently in those bones concerning which anatomists hold slightly different opinions as to the exact date at which the secondary centres develop. The lower ends of tibia, ulna, and the upper end of the radius are the most notable examples. Possibly this difference in the date of appearance of the centre depends upon slight variations in the relative proportion of bone and cartilage.

The rule also explains why the centre for the lower end of the fibula should appear before that of the upper end, being the reverse of the order in which they appear for the extremities of the tibia, but the ratios of cartilage and shaft are in the reverse order also, thus affording an admirable example of application of "The Rule of Ratios."

## NOTES ON THREE CASES OF CEREBELLAR DISEASE.

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IN vol. xv. of this *Journal* I published in detail the history of two cases of cerebellar disease, in regard to one of which I ventured to express the opinion that the patient would become a general paralytic. That opinion has been realised. Symptoms of insanity set in a few months ago, and the patient is now an inmate of our Borough Asylum. The other case was one of tubercular disease, which had involved the whole of the left lobe of the cerebellum, without giving rise to any of the usual symptoms of cerebellar disease, such as staggering or muscular inco-ordination.

In the cases which form the subject of this paper the usual symptoms which belong to disease of the organ are met with, and an additional interest is perhaps attached to them, seeing that they each belong to different periods of life. The middle lobe is the seat of the lesion in two of the patients, and the left lobe in the other.

*Case 1.*—On the 10th September 1881 I was in consultation with Dr. Hopper of Felling, when he kindly asked me to see George Garrett, a boy four years of age. Two months before this the boy had fallen on the back of his head, and on the following day it was noticed that he staggered a great deal, and that he could scarcely walk. After the accident patient continued to vomit occasionally, and complained frequently of pain in his head. Until the date of the fall there was no indication of disease so far as the parents noticed. The boy was unable to stand; he was no sooner placed on his feet than he fell backwards. The diagnosis was cerebellar disease, and, as the case was one of interest, I had him placed under my care in the Newcastle Infirmary.

On the 26th September patient was very sick, and was frequently convulsed. After this he was completely blind. Ophthalmoscopic examination revealed nothing more than marked pallor of the discs. The vessels presented nothing remarkable. When the convulsions ceased their place was taken up by small tremors, which affected the arms and legs, and those persisted more or less to the end.

In the further notes of the case I find it stated that he changes his colour repeatedly; one moment he is extremely pale, in another he is flushed. The *tâche cerebrale* is well marked. When lifted out of bed he whines; he never cries. When spoken to he simply whines, and answers "what" to every question. Placed on his feet, it is noticed that he cannot walk, or even stand, unless he is supported—that his head is carried too far back, and that there is marked opisthotonos. His legs are somewhat rigid, and patellar tendon reflex is increased. On the nurse helping him to walk, it is observed that he gets along only by performing a succession of jumps, both legs being lifted with a kind of jerk. His breathing at times is remarkably slow, not more than twelve in the minute. Both pupils are dilated, but are unequal, and they keep changing. First one is dilated, and then the other. Conjunctiva is insensitive to the touch. The head is very large, measuring  $20\frac{3}{4}$  inches, and there is a heavy bulging occipital region.

The course of the illness was downward from the first. The tremors and vomiting never left him, and he remained blind. The inability to stand and the opisthotonos remained marked to the end, before which emaciation developed and made rapid progress. The power of swallowing was in great measure lost, and he died very slowly comatose.

At the post-mortem the heart, lungs, liver, spleen, and kidneys were found healthy.

*Head.*—On removal of the scalp it was found that there was slight separation of the sutures, especially the sagittal and coronal, and, on exposing the dura mater, the brain was found soft and fluctuating. The convolutions on the upper surface of the brain were flattened, but there was no undue adhesion of the dura mater. A great quantity of fluid escaped on removal of the brain. The temporo-sphenoidal lobes were more adherent than usual. In the fissure of Sylvius there was complete absence of tubercular deposit. A considerable quantity of fluid escaped from the lateral ventricles when they were opened. The brain itself was healthy. The cerebellum exhibited a small area of white thickened membrane on the upper surface of the middle lobe. The cerebellum, as a whole, was too bulky, and the middle lobe was almost entirely occupied by a soft gelatinoid growth, which, on microscopical examination, proved to be a glioma. The other lobes were healthy.

*Case 2.*—James Wilson, aged eighteen years, an errand-boy, was admitted into the Newcastle Infirmary on the 4th October 1882, complaining of pain in the back of his head, neck, and eyeballs. Patient, who is a well-developed lad, was healthy until about three years ago, when he fell off a swing on the back of his head, and although the distance was only a few feet, he was stunned, and remained unconscious for a short time. He was able to walk home, but about a week after the injury he was obliged to keep in bed, as he was suffering from very severe pain in the crown of his head, and vomiting, and from numbness in his face and tongue. There was no disturbance of sight, and the case was considered by a medical man who saw him to be one of biliousness. From this he recovered. A little more than

two years ago he had numbness in one of his legs and one of the sides of his face, which one, however, he cannot say. This attack passed off, and, with the exception of slight headache, which had remained after the original injury, patient remained remarkably well until three weeks before his admission. After carrying a heavy weight on his head one day, he suffered from severe headache and sleeplessness. Thereafter he began to experience giddiness, and had a tendency to fall forwards, or to the left. He is short-sighted, as he calls it, and attributes it to the original injury.

Pain in the head, especially over the occipital region, is now a constant symptom. It is very severe, and usually brings on vomiting. Both discs are extremely pale, the pallor amounting to a hard chalky whiteness: their border is thickened and dark, the vessels in both are obscured in their course, and are unequal in their mode of distribution. At the point at which the veins pierce the disc, the lower and internal vessel is ill-defined, blunted in outline, and has the appearance of a white film surrounding it. Patellar tendon reflex is increased, especially in left leg; ankle clonus absent; plantar reflex not appreciably altered. The muscles of both legs are well developed, and there is no paralysis. When asked to walk, it is noticed that the patient staggers slightly from side to side; he can turn round pretty well, and can stand or walk with his eyes closed. The dizziness of which he complains is not increased by closure of the eyes. When lying in bed he is comparatively free of pain; it is only when effort is made that he complains of headache and giddiness.

Thoracic and abdominal organs are healthy. Was ordered mist. pot. brom.

Under this line of treatment patient improved in his gait, and seemed to be doing remarkably well in every respect (with the exception of the headache), when, on the morning of October 18, he died somewhat suddenly. For the last two or three days the pain had been very severe, especially in the back of his head, and the vomiting had been rather frequent, but with all this he was very cheerful. On the afternoon of the 17th October he was visited by some of his friends, to whom he seemed remarkably well. Towards evening the pain in the head became very severe, and it increased as time went on. In a paroxysm of pain he screamed, was seen to put his hand to his head, and fall back dead.

*Post-mortem.* — Calvaria presents rather a short antero-posterior diameter. There is flattening of the convolutions of both hemispheres. As the brain lay on its superior surface the anterior portions of the temporo-sphenoidal lobes bulged. Vessels at base healthy. Lateral ventricles contain large quantity of a clear fluid like water. The left corpus dentatum in cerebellum is unusually soft; the tissues there being diffuent, the softening extends from the left corpus dentatum into the right side of cerebellum, affecting a portion of the right hemisphere, but leaving its corpus dentatum normal. The fourth ventricle is dilated; its floor is unusually soft. The sinuses contain a large quantity of blood, and there is a large quantity of fluid in the spinal

canal. In the left cerebellar lobe, which was softened, a small reddish grey patch was detected, which, under the microscope, was found to be composed of numerous large round cells without any stroma, and, consequently, a sarcoma.

The third case is one of exceptional interest, and is, if the diagnosis of cerebellar hæmorrhage is correct, one of very great rarity. For the opportunity of seeing it I am indebted to my friend Dr. Strang, of Newcastle, who kindly asked me to see the case, and to make use of it in any way I pleased.

*Case 3.*—G. H., aged thirty-four years, a joiner, was seen by us in the early part of October 1882, at which time we found him suffering from loss of speech, inability to stand or walk unless supported, extreme muscular inco-ordination of arms and legs, and a tendency to fall backwards. It appears that one evening, something like eighteen months ago, patient, when engaged in a game of dominoes, suddenly lost the power of speech, and along with that his left arm and leg became powerless. He did not lose consciousness. His speech was so unintelligible that he could not be understood, and he himself was aware of its indistinctness. His friends helped him home, and after being ill for about ten weeks, he so far recovered that he was able to go about, although there still remained some weakness in his arm and leg, and slight indistinctness in his speech.

Two weeks ago patient was in the country, and just as he was sitting down to breakfast he complained of very severe headache. All at once his speech became affected, and vomiting immediately set in. He was not convulsed, nor did he lose consciousness, but it was noticed that he could neither stand nor walk. The first time we saw him his speech was so indistinct that he could not be understood. The patient was perfectly sensible, and knew everything that was said to him. There was no loss of power or sensation in his arms or legs, no opisthotonos, no giddiness; he was sleeping well at night, and had a good appetite. There was no paralysis of the face; the tongue was clean, and pupils were slightly dilated. Patellar tendon reflex was increased in both legs. He could not stand unless he was supported, and he would have fallen when his eyes were closed. On getting him to walk between two supports it was noticed that his legs were lifted high and awkwardly, that they were thrown outwards and downwards with too much force. Before getting him out of bed it was noticed that the movements of the arms were too extensive for what was required. There was no urinary or rectal trouble. Heart's sounds healthy; second somewhat accentuated.

Five or six weeks after my first visit I saw this patient again. He was sitting on a chair at the side of the fire. His speech had considerably improved under the use of a mixture of potassium iodide. There was no paralysis, but he could neither stand nor walk unless he was supported. As the result of his awkward efforts to get into his chair he had almost broken it. With a grip of the mantelpiece he



could move a yard or two from his chair, but over his feet and legs he had no control; they were thrown about just as by one who is suffering from locomotor ataxia. I was now able to understand a good deal of what he said to me. In answer to my inquiry, he admitted having had syphilis eight years ago. His emotional faculties seemed to have undergone a change since the last time I saw him. The slightest thing would make him cry.

While the function of the cerebellum is in great part unknown, physiology and pathology alike proclaim it the central organ of co-ordination, as "that part of the central mechanism by which external impressions are immediately co-ordinated with certain responsive actions." The cerebellum then is regarded rather as the regulator than the initiator of muscular movements. Wherever the effects of cerebellar disease have been noted, it has always been in the direction of disturbed muscular adjustment, and it is because of their bearing upon this point that I have thought the record of three cases of cerebellar disease might be of some interest.

Setting aside the question of the nature of the pathological conditions in the case last of all reported, and which are doubtless the result of syphilis, we have evidence in these three cases that lesion of the middle lobe of the cerebellum has been more destructive than lesion of a lateral lobe. From this fact, however, no conclusion is drawn, for in cerebellar as in cerebral disease, effects vary according to the rapidity with which the disease is developed, and the nature of the lesion, that is to say, whether it excites or destroys the tissues. The cerebellum, for instance, has been divided in the middle line in an antero-posterior direction, and there have been no disturbances of equilibrium of any importance noticed (Ferrier), and the whole of a cerebellar lobe has been destroyed, as in one of the cases I previously reported in this *Journal*, without there being any disturbance of co-ordination whatever. It is the exception, however, to find lesions in any of those areas which are not followed by symptoms. The cases just reported strongly support the opinions based upon experimental research, particularly the first and third. In each of these the middle lobe is the seat of disease: in one of them the head was markedly retracted, and in both the tendency to fall backwards was very noticeable. Nor in the other is there

wanting the well known sign of lateral disease. In it—the second case—though the staggering was not extreme, yet it was a prominent symptom, and that too in the absence, as shown after death, of anything like an extensive lesion. The lesion was not extensive, but its depth in the lobe raises the question as to whether there is not something in Vulpian's statement that lesions affecting the deeper parts of the cerebellar lobes are followed by greater disturbance of equilibration than the more superficial. It raises also another point, and it is this, whether, in the case of disease affecting one of the two lateral lobes of the cerebellum functional activity is not relegated to the other, as some suggest, or whether there is not the acquisition of a power by the individual through the medium of his corpora striata, whereby he is able to co-ordinate his muscles—the acts coming probably more within the range of consciousness—so that what was lost by cerebellum is gained by cerebrum.

The suddenness of the symptoms in the third case is of very great interest. This fact, with the history of syphilis, and a previous cerebral affection, points to the lesion being in all probability hæmorrhagic. We know the tendency which syphilis has to produce disease of blood-vessels, and a form of pachymeningitis, from both of which blood may easily come. Leaving this point, however, we have a sudden illness, in which there is complete loss of ability to stand or walk, vomiting and headache, and no paralysis or loss of consciousness, these two latter being points of great importance in the diagnosis of cerebellar disease.

It is noted that in the three cases the patellar tendon reflex is increased. Possibly in the case of H. there is some descending degeneration in the lateral columns of the spinal cord, as the result of his former illness, but in all of them there is the probability of a cerebellar origin. I have no means of knowing at present whether in cerebellar disease descending degeneration has been found in that part of the spinal cord known as Flechsig's tract,—that is to say, the area immediately behind the crossed pyramidal tract in the lateral columns, and which is said to be directly continuous with the fibres of the cerebellum,—to such an extent as to influence the excitability of the adjacent areas. In the lad Wilson—the second case—the patellar tendon reflex was increased in the left leg, and after death it was the left cerebellar

lobe that was found to be the seat of disease. It has been shown by anatomists that fibres pass from the left half of the cerebellum to the right cerebral lobe, and that other cerebellar fibres pass directly downwards, through the inferior peduncles, to meet those coming from the right cerebral lobe before they decussate, so that a very intimate relationship exists in the motor channels of the spinal cord between cerebral and cerebellar fibres. Either then a degree of descending degeneration is developed in the spinal cord in cerebellar, as in some forms of cerebral disease, or by means of the relationship just shown there is inhibition of the functional activity of the paths by which voluntary impulses travel.

The lad Wilson exhibited no tendency to fall when his eyes were closed, a fact of some importance when we know how important visual impressions are in the mechanism of equilibration. Though he complained of short-sightedness, yet his sight was good. Both discs exhibited signs of advanced degeneration; they were extremely pale; their arteries were attenuated, and the veins distorted—an atrophied condition the result of increased venous pressure in the first instance. It is noteworthy that, considering the amount of atrophy met with in optic discs, the sight is in many cases of intracranial disease very little affected.

**A CONTRIBUTION TO THE ANATOMY OF THE INDIAN ELEPHANT.** By R. J. ANDERSON, M.A., M.D., *Demonstrator of Anatomy, Queen's College, Belfast.*

THE very complete account of the anatomy of the Indian elephant, published in vols. xii. and xiii. of the *Journal of Anatomy and Physiology*, leaves little to be desired. An opportunity has been afforded me of examining a specimen of this animal, and in almost all points I have been able to verify the descriptions of Messrs. Miall and Greenwood.

In some points, however, my specimen differs from that of the above-mentioned anatomists. In a few others the description is supplemented. Where I do not note the difference, the descriptions are nearly alike.

The cutaneous muscle of the abdomen, wide above and narrow below, arises from the subcutaneous tissue above, and is inserted into the fascia of the thigh below. The fibres are parallel to those of the rectus, and overlie the pectoralis major and rectus.

The trapezius in this specimen differs from that of the specimen of Miall and Greenwood in having an origin from the inner half of the superior curved line of the occipital bone, as well as from lig. nuchæ and dorsal spines. The upper fibres pass to a tendon at the anterior edge of the muscle, which is inserted near the upper end of the spine. The posterior fibres are collected to a tendon which is inserted into the posterior border near the upper part.

The masto-humeral muscle consists of three parts. The posterior part is not mentioned in Miall and Greenwood's description, and may be regarded as a separate muscle. The posterior part arises from the basilar part of the occipital bone, is 3 inches in width and flat, and is inserted into the anterior border of the spine below, and the fascia covering the deltoid. The second part arises from the basilar portion of the occipital bone, close to the preceding, and is inserted with the third portion into the humerus in front of the deltoid. The third portion is round, and takes origin from the mastoid process. A

tendinous inscription occurs in the second and third parts, near the shoulder.

The sterno-maxillaris consists of three pieces below and two above. The middle part arises from the sternum, the lateral from the first rib, and these form a single muscle at the middle of the neck, which divides above into two muscle bands that are inserted into the inferior maxilla. Miall and Greenwood give this muscle as arising from the first rib, but state that the condition of their specimen did not enable them to note the attachment accurately.

The sterno-mastoid arises from the sternum for 3 inches, and is inserted into the lower part of the zygoma. Miall and Greenwood give the origin from the first rib. Mr. Young, in vol. xiv. of this *Journal*, gives the attachment to the sternum.

The rectus capitis posticus major and minor correspond to Miall and Greenwood's description, but I find no trace of a lateral rectus.

As in Mr. Young's specimen, the pectoralis major has a very extensive origin, from the sternum and ext. oblique aponeurosis. The upper fourth is superficial, and, as in man, passes down in front of the lower. The insertion is internal to that of the masto-humeral and outside the biceps, and into the fascia of the arm.

A triangular muscle, described as pectoralis minor by Young, I find arising from the cartilage of first rib, and inserted into the fascia covering the supra-spinatus. But no muscle comparable to a pectoralis minor inserted into the upper end of the humerus is present.

The deltoid in my specimen has a more extensive origin than that of Miall and Greenwood. It arises from the posterior border of the spine, the unciform process, posterior border of scapula (origin, fleshy below, tendinous above), and the inter-muscular septa between it and the infra-spinatus and triceps.

The biceps has a single head of origin from the upper part of the glenoid process. It represents the gleno-radialis of Krause. The coraco-brachialis, representing the coraco-ulnaris of this anatomist, arises from the coracoid process and capsulo, and is inserted in the inner border of the humerus in its whole length. (This agrees with Mr. Young's description.)

The pronator teres has a large triangular part, which underlies the long fibrous band, and has a fleshy attachment for 5 inches. (More extensive than in Miall and Greenwood's specimen.)

A radio-carpus, not described by the above-mentioned anatomists, arises from the surface of the radius external to the pronator teres extending to the lower extremity, and from the interosseous membrane. The fleshy part of the muscle is succeeded by a flat tendon that is inserted into the anterior ligament of the wrist joint. The origin of the muscle corresponds nearly to that of the flex. long. pollicis in man.

The tensor vaginæ femoris in this specimen has a more extensive insertion than that described by Miall and Greenwood. Arising from the anterior third of the iliac crest and fascia lata, it is inserted into the fascia lata above the middle of the thigh, half the circumference of which it surrounds. It is connected with the gluteus maximus and biceps behind.

The internal oblique of the abdomen arises from the anterior fourth of the iliac crest and the outer part of Poupart's ligament, the fascia, lumborum, and last rib but two, and the last but three by tendinous slips. The fibres radiate so as to form a fan-like muscle. It is inserted into the abdominal aponeurosis. A flat muscle band is inserted into the last rib behind.

The transversalis abdominis arises from two-thirds of the iliac crest, fifteen ribs, and the lumbar aponeurosis. The lower margin of the muscle forms an arch 10 inches in length above or in front of Poupart's ligament.

No sartorius is present.

The tibialis posticus arises from the posterior surface of the tibia below the popliteus, the fibula and septa, and is inserted into the 2nd, 3rd, and 4th metatarsal bones.

The following are the measurements of the viscera:—

The stomach is 1 metre 40 cm. long, 33 cm. in diameter.

The cardiac opening is 44 cm. to the right of large end.

Length of small intestines, 60 feet.

Length of large intestines, including cæcum and rectum, 26 feet.

Length of cæcum, 2 feet 2 inches.

Circumference of cæcum, 50 inches.

Circumference of colon: 1st part, 34 inches; 2nd part, 30 inches.

Rectum, 14 inches.

Small intestine, near stomach, 7 inches; near cæcum, 10 inches.

*The spleen* is attached to the stomach by the gastro-splenec omentum. Its weight is 3 lbs. 13 ounces. Length, 1 metre 24 cm.; breadth at widest part, 10 cm.

The liver has two lobes, weighs 30 lbs., is 69 cm. long, 39 cm. broad, and 7 cm. thick.

The pancreas weighs  $3\frac{1}{4}$  lbs.

The heart, 15 lbs. (a portion of the aorta is included).

The lungs, 32 lbs. 4 oz.

The brain,  $6\frac{1}{2}$  lbs.

The kidneys: The right—length,  $26\frac{1}{2}$  cm.; breadth,  $19\frac{1}{2}$  cm.; thickness,  $5\frac{1}{2}$  cm. The left—length, 29 cm.; breadth, 20 cm.; thickness, 6 cm. The weight of the right is  $3\frac{3}{4}$  lbs., and the left 4 lbs. Eight lobes are present in each.

The bladder measures 30 cm. from base to apex, and 24 cm. in diameter transverse.

The supra-renal capsules weigh 3 ounces each; each has a double cornu internally.

*The uterus* is 16 cm. in length, and is 6 cm. in circumference.

The Fallopian tubes, each 45 cm. in length.

The vagina, 30 cm. length, 18 cm. circumference.

The genito-urinary passage, 80 cm. length, 19 cm. circumference below, 30 cm. above.

The length of the clitoris is 45 cm.

The weight of the animal was  $1\frac{1}{2}$  tons (?).

**NOTE ON A SPECIMEN OF ABSENCE OF THE PARTS  
DEVELOPED FROM THE FIRST VISCERAL ARCH  
ON ONE SIDE IN A LAMB (SEMI-AGNATHIA OR  
SYNOTIA).** By FREDERICS EVE, F.R.C.S., *Pathological  
Curator of the Royal College of Surgeons, England, and  
Surgical Registrar to St. Bartholomew's Hospital.*

THE most striking feature of the malformation consists in the exposure to view of the cavity of the mouth, owing to the absence of the right superior and inferior maxillary bones. Sloping from the posterior end of the exposed buccal cavity is a smooth groove, which is lined with mucous membrane and extends upwards, backwards, and outwards to the pinna of the ear. This groove, probably, represents the posterior extremity of the first visceral cleft, in which, under normal conditions, the external auditory meatus and Eustachian tube would have been formed.

The tongue is well developed, but is tightly tied down to the floor of the mouth on the right side.

The skull denuded of the soft parts by maceration, presents many peculiarities. All the bones, developed from, or in connection with, the first visceral arch, are absent:—viz., the inferior maxillary bone, developed from Meckel's cartilage; the palate and pterygoid bones, formed respectively in the anterior and posterior portions of the superior maxillary process of the first arch; and the superior maxillary bone itself, subsequently formed in membrane; the malleus;—all are completely wanting.

While the premaxillary bones and the vomer, which are developed in connection with the prenasal cartilage, are well formed. The right lachrymal and turbinated bones are also normal.

Some other bones of the right side of the skull and face are either wanting, ill-developed, or modified in form; but these are either directly connected in their development with some portion of the first visceral arch, or serve as attachments for some of the masticatory muscles; or they are modified in order to adapt



themselves to changes caused by the absence or ill-development of neighbouring bones.

The temporal bone is dwarfed. The tympanic portion is very small, its bulla is rudimentary, and the auditory process is altogether absent.

The petrotic bone is more largely developed. The squamosal bone extends only half the usual distance in an upward direction, but is prolonged forwards and inwards, so as in great part to fill up the portion of the base of the skull normally occupied by the right half of the basi-sphenoid, which is absent. No trace of any of the bones of the internal ear existed.

All the parts entering into the formation of the zygomatic arch and its buttresses are wanting, and owing to the absence of the malar bone and the external orbital process of the frontal bone the floor and outer wall of the orbit are incomplete.

The parietal bone also exhibits some peculiarities, which were probably secondary to the abnormalities of the temporal bone; it is larger than normal, and is divided into two nearly equal parts by a horizontal suture. At its anterior inferior angle is a small quadrate plate of bone, probably analagous to the bone sometimes found occupying the same position in human crania, and to which Professor Flower has given the name of epi-pteric bone.

The right half of the hyoid arch is complete, with the exception of the incus, the development of which Mr. W. Kitchen Parker has referred to the second arch.

The foramina for the divisions of the 5th nerve are very small on the right side; and the foramen for the carotid artery is formed in front by the sphenoid, and behind by the squamosal bone.

The bones of the left side of the skull and face are normal, except that the palatine plates do not extend to the middle line. The left half of the inferior maxilla is shortened and suddenly incurved at its anterior extremity.

The brain was well formed, and its two halves were symmetrical. All the cranial nerves existed, but the 5th and 9th nerves off the right side were small.

The preservation of the soft parts for a museum specimen<sup>1</sup>

<sup>1</sup> The skull and soft parts are preserved in the Royal College of Surgeons' Museum, Teratological Series, Nos. 193b and 193c.

did not permit of a minute dissection of the blood-vessels, the condition of which was, therefore, undetermined.

The lamb was in other respects well formed, and of the usual size. It had been observed to suckle, and appeared in good health when it was killed on the day after its birth.

*Remarks.*—The chief interest of this abnormality consists in the remarkable manner in which the parts developed from the first visceral arch are mapped out by their absence on one side.

The specimen, perhaps, also affords, in some measure, a proof of the correctness of the received descriptions of the mode of development of some of the bones of the face and skull.

It presents another point of interest in the manner in which the inter-dependence of co-related structures in their development, is illustrated; as by the modification of the parietal and squamosal bones to fill up the gap in the skull, which would otherwise have been occasioned by the absence of the greater part of the right half of the basi-sphenoid; and by the absence of the zygomatic arch, the existence of which as a muscular attachment was rendered unnecessary.

As regards the cause of the malformation only negative evidence is gained, from the fact that the brain and cranial nerves were well developed. The malformation may have been due to some disease of the blood-vessel, causing thrombosis at an early period of fœtal life. To such causes Kundrat<sup>1</sup> refers some congenital defects of the brain substance. This explanation, however, is rendered improbable by the common cases in which absence of the maxillary bones on both sides has been observed, especially in lambs.

The rarity of the condition described is attested by the fact that no case of semi-agnathia is mentioned in the standard works on malformation by Geoffroy St. Hilaire, Gurlt, Förster, and Ahlfeld.

<sup>1</sup> *Die Porencephalie*, Leuschner and Lubensky Graz, 1882.

THE OSSIFICATION OF THE TEMPORAL BONE. By J.  
B. SUTTON, *Senior Demonstrator of Anatomy at the Middlesex  
Hospital.* (PLATE XVIII.)

IN his *Lectures on the Elements of Comparative Anatomy*, published in 1864, Professor Huxley described the "pars petrosa" of the human temporal bone as arising from three independent centres of ossification. He distinguishes an *opisthotic* centre, which appears near the fenestra ovalis, and ultimately gives rise to the promontory, lower portion of cochlea, surrounds the fenestra rotunda, and contributes half the contour of the fenestra ovalis; it also gives rise to the carotid canal by developing a lamella of bone, which gradually wraps itself around the carotid artery, and so converts the primitive groove for that vessel into a complete tube, at the same time furnishing the inner part of its floor to the tympanum. The second centre is termed *Pro-otic*. It is situated at the outer end of the superior vertical semicircular canal. It invests the roof of the cochlea, the superior, and part of the posterior vertical semi-circular canals, the internal auditory meatus, and forms the tegmen tympani. To it is also due the upper half of the fenestra ovalis, and a considerable portion of the pars mastoidea.

The third centre gives rise to the mastoid process, and invests the posterior part of the posterior semicircular canal. This nucleus is named the *Epiotic*.

These centres begin to appear after the middle of the fifth month, increase rapidly in the sixth month, at which date nothing is met with but a beautiful reticulated cartilage ossification, which later is absorbed and replaced by true vascular bone. The modiolus and lamina spiralis ossify at the end of foetal life without ever having been cartilaginous. The foregoing account has been generally adopted by anatomists in this country, but Kölliker in his *Entwicklungsgeschichte*, 1876-79, gives countenance to the views of Vrolik, who describes the origin of the petrous bone from four centres, with two additional ones for the mastoid portion.

The situation of these centres, according to Vrolik, is as follows :—

- (a) One on the promontory corresponding to Huxley's opisthotic.
- (b) Another in the neighbourhood of the aqueductus vestibuli.
- (c) A third on the roof of the internal auditory meatus, representing the pro-otic.
- (d) The fourth gives rise to the roof of the cochlea.
- (e) Lastly, two nuclei, the representatives of the epiotic, give origin to the mastoid process.

Recently I have investigated the mode of development of this important bone, particularly with regard to the relation borne by the various ossific centres to the nerves found grooving the bone in the adult. Before giving an account of my observations on the ossification of the petrous bone, I will give a description of the periotic cartilage and the parts in relation with it in their early cartilaginous conditions :—

At the fifth month of intra-uterine life the squamo-zygomatic plate and the tympanic annulus are already ossified, but the labyrinth, malleus, incus with the orbicular element, and the stapes are in the primitive cartilaginous condition. The Folian process of the malleus and the Reichertian cartilage are well seen, as two rods embracing the tympanic bone (see fig. 1, *sty. hyal*).

The anterior extremity of the future pars petrosa is smoothly rounded, corresponding exactly to the first turn of the cochlea, and the fenestra rotunda opens on the under surface of the skull. There is no canal for the carotid artery, no Eustachian tube, or the least evidence of pyramids, anterior or posterior, for the tensor tympani and stapedius muscles respectively.

The three semicircular canals may be clearly traced by the prominences they form on the surface of the cartilage. The superior vertical canal is especially evident, on account of the large fossa<sup>1</sup> which it surrounds being exposed when the dura mater is stripped off. Posteriorly to the semicircular canals is a broad plate of cartilage, in which the ex-occipital may be seen developing (*ex. oc.*, fig. 1), for at this date there is no line of

<sup>1</sup> Floccular fossa.

demarcation between the temporal and occipital portions of the cartilaginous cranium. A hole in the cartilage behind the eminence formed by the posterior canal, marked *j* in fig. 1, transmits a vein from the interior of the cranium to join the external jugular vein. This foramen is represented in the osseous skull by the opening immediately posterior to the mastoid process, which affords passage to an emissary vein from the lateral sinus, frequently of considerable size.

Above the external semicircular canal, and jutting out at right angles to the main portion of the ear capsule, is a prominent ledge, which extends as far forwards as the geniculate ganglion of the facial nerve, a spot corresponding to the hiatus Fallopii in the adult bone. In contact with the under surface of this shelf, and lodged in a recess excavated in the cartilage, will be seen the malleus and incus, their outer surfaces lying in contact with the squamosal and above the *membrana tympani*. Although the *membrana* is present, there is as yet no tympanic cavity. This ledge of cartilage (for reasons to be presently assigned) should be termed the *pterotic ridge*, later it becomes transformed into the *tegmen tympani*, or roof of the *tympanum* (*Pt. r.*, fig. 1).

At this stage the Fallopian canal, which in the adult runs so singular a course, has no existence except as a canal barely one-sixteenth of an inch long, extending from the internal auditory meatus to the position of the future hiatus Fallopii. From this point the seventh nerve (*portio dura*) lies in an open groove, passing backwards between the incus and the labyrinth directly above the fenestra ovalis, finally coursing downwards to gain the interval between the future styloid process and the mastoid portion. Thus, from the spot where the nerve develops the geniculate ganglion its course is extra-cranial, as has been pointed out by Gegenbaur.<sup>1</sup>

The *chorda tympani*, which later runs so complicated a course, merely passes between the tympanic ring and the cartilaginous capsule to gain the lingual branch of the fifth, without traversing any canal. At this date, and even as late as the sixth month, the palatine branch (great superficial petrosal) of the facial lies outside the cranial cavity, for the floor of the skull between the

<sup>1</sup> *Morph. Jahrbuch*, Bd. ii., 1876, "Bemerkungen über den canalis Fallopii."

rounded anterior extremity of the periotic cartilage and the alisphenoid is formed only by dura mater. It is only when the roof of the tympanum and the anterior pyramid are ossified that this nerve can be said to lie inside the cranium. Even in the adult it becomes a question whether structures situated between the bone and cranial dura mater, and not in any way perforating it, can be correctly spoken of as intercranial, that is, from a morphological point of view. In the case of the palatine nerve this question is an important one. The fact of its being, for a certain<sup>1</sup> period at least, outside the cranium, supports strongly the view held by certain morphologists that the palatine nerve of the seventh, which in fishes, particularly elasmobranchs, is altogether outside the cranium, is homologous with the great superficial petrosal of Mammalia.<sup>2</sup> In the embryonic condition this nerve is relatively far larger than in the adult state.

The tympanic branch of the glossopharyngeal (Jacobson's) crosses the promontory and passes to its destination without traversing canals or foramina of any description.

Even the auricular branch of the pneumogastric (Arnold's) crosses the small elevation in the cartilage, representing the future mastoid process, communicates with the portio dura (seventh), and passes to its destination behind the pinna without entering a foramen or complicated tunnel, but lies exposed in a furrow.

Consequently, the intricate course pursued by these nerves in the adult skull results from the extensive modification the parts undergo during the process of ossification. The long tunnel, which in the thoroughly ossified cranium lodges the auricular branch of the vagus, is at first merely a narrow chink left between the tympanic ring and the periotic bone, its length depending upon the large external elongation the tympanic bone undergoes after birth.

The relation of the nerves mentioned above to one another, and to the periotic cartilage and its appendicular elements, is shown in fig. 1, where appropriate letters refer to individual

<sup>1</sup> These remarks are equally applicable to the Gasserian ganglion and its three great divisions at their immediate origin.

<sup>2</sup> Consult more particularly on this point Jackson and Clarke on "The nerves of *Echinorhinus spinosus*" in the *Jour. Anat. and Phys.*, vol. x., 1875 and 1876.

nerves, the tympanic bone being dotted, so as not in any way to complicate the figure.

Ossification commences about the end of the fifth month by a small nodule which makes its appearance on the promontory (the bulging which marks the first turn of the cochlea); it quickly surrounds the fenestra rotunda from above downwards,<sup>1</sup> and spreads backwards, forming the floor of vestibule and lower part of fenestra ovalis and the internal auditory meatus respectively. Anteriorly ossification proceeds from this centre around the cochlea, forming a distinct hook-like process, terminating at the internal auditory meatus. Later, the floor of the tympanum and canal for the internal carotid artery arise as an outgrowth from the lower part of this centre, which is called the *opisthotic*.

A nodule of bone deposited on the surface of the cartilage, directly posterior to the internal auditory meatus, is termed the *pro-otic* centre. From it arises in due course the roof of vestibule, part of the fenestra ovalis, and the bony covering of the superior vertical semicircular canal; anteriorly it passes over the facial and auditory nerves, and fuses with the hook-like process of the opisthotic to complete the internal auditory meatus. *Hence the foramen of exit of the seventh cranial nerve marks the point of union of the pro and opisthotic centres.*

Almost simultaneous with the appearance of the pro-otic centre, ossific granules make their appearance over the outer limb of the horizontal semicircular canal, extending thence outwards into the pterotic ridge (tegmen tympani) and downwards to form an osseous covering to the external canal, finally blending with the opisthotic. Even in a fœtus six and a half months old the rectangular plate of bone representing this centre is easily separated, except at the angle where it joins the pro-otic portion, and even this line of junction is frequently perforated. This centre should be termed the *pterotic*.

Lastly, a nucleus termed the *epiotic* appears on the posterior portion of the posterior semicircular canal, eventually covering in this canal, and giving rise to the mastoid portion. Rarely a

<sup>1</sup> When the opisthotic first ossifies, the fenestra rotunda opens on the base of the skull, but when the floor of the tympanum extends backwards a narrow canal forms, known later as the aqueductus cochleæ, which lodges a membranous tube, with a blind end, known as the ductus perilymphaticus.

second smaller nodule may appear between the primary centre and the squamosal.

The subsequent course of events is simple. The various centres grow rapidly, and very quickly fuse, disguising all trace of their original individuality. The open groove for the portio dura becomes covered in by the pterotic growing over it to ankylose with the pro-otic and opisthotic, thus shutting off the nerve from the tympanic cavity by only a very thin plate of bone. This osseous layer encloses the stapedius muscle in a bony compartment known as the posterior pyramid, leaving a small aperture for the tendon to pass forwards to the stapes. A little chink, known as the "iter chordæ posterius," allows the passage of the chorda tympani. Below the origin of this nerve from the seventh, the stylo-mastoid foramen, or termination of the aqueduct, is completed by the union of the post-auditory process of the squamosal with the pterotic posteriorly, and tympanic bone anteriorly.

The pterotic growing forwards, surrounds the geniculate ganglion to join the portion of bone anterior to it, thus leaving this ganglion free on the floor of the skull, so that even at birth the hiatus Fallopii is in reality a fossa, and not a foramen, its complete investment not taking place until some months after birth. When the pterotic joins the opisthotic, a thin lamella of bone envelops the tensor tympani muscle forming the anterior pyramid, leaving merely a small foramen at the apex for the tendon to gain the malleus. The line of junction of opisthotic and epiotic portions is accurately indicated by the little groove lodging the palatine nerve (great superficial petrosal nerve), as it runs forward to gain the Vidian canal. Some notion of the rapidity with which the opisthotic, pro-otic, and pterotic centres develop, may be gathered from the fact that at the middle of the fifth month there is not the least trace of ossification in the periotic cartilage, but by the end of the sixth month the three portions have fused, leaving scarcely a trace of the lines of junction between their ossific centres. This fact renders necessary much patience and care in procuring and examining specimens. It unfortunately happens that during their early development the bones of the skull-base are enclosed in dense fibrous membrane, which adheres firmly to them, so that unless care is exercised delicate



pieces are very easily detached. My plan has been to macerate the skull and gently wash away the débris day by day.

The separate centres for the petro-mastoid bone and the parts developed from them may be arranged in a tabular form :—

Opisthotic (in intimate relation with the cochlea).	Cochlea Fenestra rotunda. The anterior and inferior portion of the internal auditory meatus, part of fenestra ovalis. Later, the carotid canal and floor of tympanum, and the anterior pyramid for the tensor tympani muscle.
Pro-otic. . . . .	Bone covering the superior semicircular canal, roof and posterior boundary of the internal auditory meatus, part of vestibule and fenestra ovalis and part of mastoid process.
Pterotic. . . . .	Covering for external semicircular canal, tegmen tympani, Fallopiian canal, and posterior pyramid for the stapedius muscle.
Epiotic. . . . .	Mastoid process and covering for posterior semicircular canal.

From this table it will be readily seen that the details of the mode of ossification of the complex pars petrosa, according to my observations, differs from those described by Professor Huxley in two important particulars. Briefly they may be summed up :—

- (a) The cochlea is ossified from opisthotic only. Professor Huxley says—part opisthotic, and in part pro-otic.
- (b) The parts described by me as originating from the *pterotic* centre, according to Professor Huxley, are ascribed to the pro-otic. It seems to my mind that J. F. Meckel<sup>1</sup> has some doubts concerning the true origin of the tegmen tympani and the covering of the horizontal semicircular canal from the nucleus, since termed by Professor Huxley pro-otic, although he states he could not find a particular nucleus for it.

<sup>1</sup> *Manuel d'Anatomie*, tom. ii., French translation.

His account is after this fashion :—"The horizontal semicircular canal commences to ossify at the fifth month. At this epoch the ossous piece which forms the superior semicircular canal extends backwards, downwards, and outwards, around the membranous horizontal canal. It has been impossible for me, at least, to discover a particular nucleus for this canal, which seems to ossify from an extension of the two previous nuclei."<sup>1</sup> Even Professor Huxley, in the "Lectures" referred to at the commencement of this paper, in speaking on this subject (page 153), states :—"A second very small quadrate ossification is situated at the outer end of the superior vertical semicircular canal, and *apparently*<sup>2</sup> extends into the cartilaginous *tegmen tympani*."

- (c) The remaining point of difference concerns the *epiotic*, which is described as originating from a simple nucleus, its occasional duplicity not being noted by anatomical writers previous to Vrolik.
- (d) With regard to the centre for the roof of the cochlea, described by Vrolik, I can only state that I have failed to discern it in the numerous specimens examined. What evidence can be adduced to show that the centre which has been described for the roof of the tympanum should be termed *pterotic*?

Professor Parker, in his admirable *Morphology of the Skull*, writes thus :—"A remarkable feature about the ear-capsule is the formation at an early period in nearly all types of a more or less horizontal projection called the *pterotic ridge*: it is related to, and often contains part of, the horizontal semicircular canal. Where there is a tympanum this ridge overhangs the articulation of the appendicular elements of the skull. When a tympanic cavity is developed this ridge is specialised into a *tegmen tympani* or *tympanic roof*, and there is a definite scooping of the ear mass beneath this ridge."

The ridge of cartilage, which was described as standing out

<sup>1</sup> "The two previous nuclei"; this refers to the pro-otic and opisthotic.  
This word is not in italics in the original.

above the malleus and incus, as well as the ossific centre which later converts it into the bony ledge which after birth anchyloses to the squamosal, seems to me to fulfil all these conditions. The ridge in question covers part of the horizontal canal, forms the tegmen tympani, is in actual contact with the malleus and incus (appendicular elements of the skull), whose main portions occupy, not the true tympanum, but a recess, the antrum mastoidea, scooped out beneath the ledge. The long process of the incus, the handle of the malleus, and the stapes, are the only parts of the ossicula actually in the true tympanic cavity. The relation of the ridge to the portio dura is strong evidence in favour of the homology of tegmen tympani and pterotic ridge.

At birth the bone consists of three pieces—petro-mastoid, squamosal, and tympanic annulus. The styloid process remains for some time in the condition of cartilage. The carotid canal is complete, the plate of bone separating it from the tympanum being cribriform. The floccular fossa is filled by a process of dura mater, but is still very large; the Eustachian tube and tympanic cavity are filled with foetal connective-tissue. The processus gracilis of the malleus is very large, and the ossicula are almost as large in the foetus at term as they are in the adult.

The remaining parts of the temporal bone presents certain features of interest: attention must now be directed to them. The lower edge of the squamosal is at first nearly straight, but near the end of intra-uterine life it sends a curved process downwards between the tympanic piece and the petrous bone. This curved process corresponds, as Professor Huxley has shown, to the margo-tympanicus or post-auditory process of the adult temporal bone. It is through the intervention of this process that the tympanic bone does not anchylose with the pterotic ossification. The line of fusion of the squamosal, with the tegmen tympani, is visible even in adult skulls as a fissure named petro-squamous. The tympanic piece remains as a delicate horseshoe-shaped bone up to the middle of the eighth month of intra-uterine life. After this period the upper extremities anchylose with the lower border of the squamosal; directly after, and occasionally even before birth, the tympanic piece also fuses with the sides and floor of the tympanum and

with the *processus gracilis* of the malleus, which, a few years after birth, merely exists as a stunted process, with a small bundle of fibrous tissue attached, the representative of the so-called *laxator tympani* muscle.

At birth the *antrum mastoidea* is of large size, excelling in this respect the cavity of the true tympanum. This is, of course, explained in part by the relatively large size of the auditory ossicula; the external semicircular canal forms a prominence on its inner wall. If the *stylo-mastoid foramen* be examined in a macerated specimen of the temporal bone obtained from a foetus at birth, it will be found very large, and in some instances the cavity of the posterior pyramid actually opens on the outer wall of the skull. To the anterior aspect of the foramen a tiny nodule of bone is frequently found, but so loosely fixed that a slight force detaches it. This nodule is the *tympano-hyal* of Professor Flower.<sup>1</sup> In the recent condition it is surrounded by the cartilage representing the future *styloid process*, whose proper nucleus, the *stylo-hyal*, appears later, but remains movable on the *tympano-hyal* for some years, but eventually they *anchylose*, so that in the adult all traces of the original separation of these two parts is lost. For a long time after birth cartilaginous tracts exist in the temporal bone, the most important being one between the post-auditory process of the *squamosal* and the mastoid, sometimes extending into the situation of the future mastoid cells. Another is situated between the temporal and its junction with the *ex-occipital*.

In conclusion, I would remark that if the *tegmen tympani* be accepted as the homologue of the *pterotic element* in the skull of the fish, and certainly from its anatomical relations there is no valid reason why it should not be thus recognised, then grave doubts come across the mind as to the claim of the bone resting immediately above the quadrate in the skull of the crocodile to be called "*squamosal*." If Professor Parker's views and descriptions regarding the *pterotic ridge* be held correct, and to my mind they undoubtedly are, then the *squamosal* of the crocodile's skull is not homologous with that bone in mammals, but with the "*tegmen tympani*." Of course the crucial test would be as to its mode of origin, *cartilage* or *membrane*, but

<sup>1</sup> *Osteology of the Mammalia.*

unfortunately very young crocodiles are not always at hand; nevertheless, in the adult skull it fulfils all the anatomical conditions required of the pterotic, more especially since it has been settled, on fairly secure grounds, that the malleus is the homologue in the mammalian skull of the quadrate bone of animals lower in the scale. This view is rendered still more probable by the fact that, when Professor Huxley first published his lectures on this subject, he described the bone forming part of the articular facet for the hyomandibular in the skull of the pike as the squamosal, though he hinted in a footnote that possibly it might turn out to be the pterotic; later he came to a fresh determination of its nature, and changed its name to pterotic. If any one takes the trouble to investigate the relations of this bone in the skull of the pike, and compare it with the bone lying above the appendicular elements of the skull of the crocodile, named the squamosal, and compare it with the tegmen tympani of a mammal's skull, he will, I think (unless it can be clearly proved to originate from membrane), be convinced that the bone now called squamosal in the crocodile's skull is in reality the pterotic.

#### EXPLANATION OF PLATE XVIII.

Fig. 1 ( $\times \frac{2}{3}$ ). The periotic cartilage and accessory parts, with the principal nerves in relation, from a human foetus at the fifth month. *Pt. R.*, Pterotic ridge slightly exaggerated.

*VII.* Portio dura giving off the great superficial petrosal (*G.Sp.P.*), a branch to the small superficial petrosal (*S.Sp.P.*), and the chorda tympani (*Ch.T.*). *Mek. C.*, Meckel's cartilage; *Ty. of IX.* tympanic branch of glossopharyngeal nerve (Jacobson's); *Sty. hyal.* styloid process. The dotted line represents the tympanic bone.

Fig. 2. Temporal region of the skull seen from within. *C.*, cochlea; *Fall. Canl.*, Fallopian Canal; *Int. Aud.*, internal auditory meatus; *M.F.*, mastoid foramen; *S.S.C.*, superior semicircular canal; *Pt. R.*, pterotic ridge; *S.O.*, squamo-occipital; *Ex. Occ.*, ex-occipital developing; *A.C.F.*, anterior condyloid foramen.

Fig. 3. The periotic cartilage as seen from the interior of the skull. To show the relation of the three centres, opisthotic, pro-otic, and pterotic, to one another. *Ep.* The black dot marks the situation where later the epiotic appears.

Fig. 4. The temporal region of the skull seen from without, showing particularly the opisthotic ossification curling round the cochlea. Other references as before. The pterotic ridge somewhat exaggerated for the sake of distinctness.

CASE OF PRIMARY EPITHELIOMA OF THE LUNG,  
WITH SECONDARY DEPOSITS IN THE KIDNEY,  
VERTEBRÆ, AND RIBS. By W. E. HOYLE, M.A.,  
M.R.C.S., F.R.S.E., *Naturalist to the "Challenger." Expedition  
Commission, and formerly Demonstrator of Anatomy in The  
Owens College Medical School.* (PLATE XIX.)

(Studies from the Pathological Laboratory of The Owens College, Manchester.<sup>1</sup>)

SOME time ago Professor Dreschfeld placed in my hands for microscopical examination portions of the affected lung of a patient who had died suddenly from pulmonary hæmorrhage in the Manchester Royal Infirmary, along with the following account of the clinical history of the case:—The patient was admitted in a generally febrile condition, but there were no symptoms of any one of the acute exanthemata; the physical examination of the chest presented no abnormal signs; great pain down the right side and back was complained of, and the patient was shortly removed to one of the surgical wards. Matters continued pretty much in this condition, and no definite diagnosis had been arrived at, when a sudden attack of profuse hæmoptysis terminated the case fatally.

The *post-mortem* examination was performed by Mr. A. H. Young, F.R.C.S., who has been good enough to furnish me with the following abstract of his report:—

Body well developed but somewhat emaciated. A small encysted hydrocele in connection with the epidermis occasioned an enlargement on the left side of the scrotum, and the inguinal glands on both groins were slightly enlarged and indurated. With these exceptions, there were no external manifestations of morbid changes. On opening the thoracic cavity the lungs were found markedly emphysematous anteriorly, and so voluminous that their anterior margins came into apposition, and entirely concealed the pericardium. Both lungs were universally pigmented. Section revealed chronic bronchitic changes, the smaller bronchial tubes being surrounded by an increased amount of peribronchitic tissue, and their lumen occupied by thick muco-pus. In the upper lobe of the left lung there was an irregular cavity about

<sup>1</sup> This investigation was completed, and the paper for the most part written, more than a year ago, during the period of the author's connection with the Owens College.

the size of a walnut. It possessed very thick, indurated, and rugged walls, and communicated by a small irregular opening with the main bronchus; this latter was surrounded with dense fibroid-like new formation, continuous with the walls of the cavity. Both bronchus, with adjoining branches, and the cavity itself, were filled with recent blood clot. Blood clot was also found in the bronchus of the right lung, whilst in the upper lobe of this lung blood had passed into the air cells themselves.

Pleuritic adhesions of old formation existed on both sides, and the visceral layer of the left pleura, especially on its mediastinal aspect, was further considerably thickened and fibroid.

Heart and pericardium were fairly normal.

*Abdomen.*—Peritoneum healthy.

Alimentary canal from mouth to anus was entirely free from molar changes.

Liver (3 lbs. 8 oz.) was congested. Capsule was marked by superficial cicatrix-like thickness. A small cavity, containing clear straw-coloured contents, was situated in the substance of the left lobe; wall of cavity, thick and fibroid, was coated internally with a thin layer of tenaceous, curdy, puriform material.

*Kidneys* (5 oz. each).—Left kidney presented four rounded nodules of new formation; their size varied, the smallest measuring half an inch in diameter, the largest 1 inch. The majority were situated deeply in the substance of the organ, one only, and that the largest, showing itself on the surface, where it formed a convex, slightly umbilicated, projection. Each nodule was soft in consistency, yellowish in colour, and distinctly circumscribed. The largest showed a puriform softening at the centre. A single small nodule, of precisely similar nature, was found in the substance of the right kidney. Ureters, bladder, and urethra normal. Spleen enlarged, capsule thickened, substance soft, but free from new formations.

*Ossæous System.*—In the right iliac fossa a large irregular cavity, with walls and contents similar to that existing in the liver, was found in relation to the lower lumbar vertebra. The bodies and laminæ of the last two of these formed the internal boundary of the cavity, and their transverse process projected therein. The surface of bones implicated was bare and carious, the projecting transverse processes appearing necrotic, whilst that of the fourth vertebra was also detached.

The third dorsal vertebra presented a rounded projection beneath the periosteum, on the right side of its body. This was due to a mass of soft whitish material of new formation, which appeared to invade and involve the bone itself. The fifth rib, near its posterior angle, was surrounded by similar material, which gave rise to a fusiform swelling, whilst the underlying bone was so far implicated that spontaneous fracture had occurred.

*Microscopical Examination.*—The organs particularly examined were the lung and bronchus, kidney, vertebræ, and the head of a rib. With respect to the method employed, it is only necessary

to say that the sections, of which a large number were cut, were made with a freezing microtome, variously stained with eosin and logwood, or alum-carmin, and mounted, some in dammar, some in Farrant's medium.

*Examination of the Lung.*—The implication of the lung by the morbid growth had proceeded so far that any attempt to discover the point of origin of the disease was quite out of the question, and I was obliged to content myself with studying the mode of its subsequent progress. I had expected that this would have been most extensive in the epithelium of the glandular structures, but to my surprise I found that it was the fibrous tissue, lying between the cartilaginous rings and the lung parenchyma, which was the part selected for its most rapid development.

Longitudinal sections of the bronchus gave me a very complete view of its mode of growth (fig. 1); and in such a section it could be seen that although the tumour had attained its mature structure in the fibrous layer above mentioned (fig. 1, *c*), yet the bronchial glands presented a perfectly normal appearance, except for a small amount of round-celled infiltration between the alveoli (fig. 1, *g*), which was to be attributed to slight peribronchitis.

On the extreme margin of the growth were situated small irregularly oval nucleated cells, lying between the connective-tissue fibres (fig. 1, *b*). A short distance inwards from these might be noticed elongated groups of somewhat similar cells, occupying fusiform spaces in the connective tissue (fig. 1 *c*). Proceeding still further from the edge of the growth, there were found cell groups gradually increasing in size and number of constituent elements, and separated by continually decreasing amounts of fibrous tissue.

It would seem to be clearly proved by these appearances that small epithelial cells penetrate into the interstices of the connective tissue, and there proliferate, thus giving rise to the fusiform groups above alluded to, and that by their growth they cause the disappearance of the fibrous tissue: it seems also that the elastic fibres persist longer than the white ones.

The cells of the tumour become eventually very large (in many cases 0.015 mm. in diameter), and although the majority of them are approximately ovoid, there are some which exhibit



considerable variety in form. There are numerous nests in the tumour, but there is nothing in their appearance to call for special description.

This mode of invasion of epithelioma of the bronchus differs from those which have been observed by other investigators. Stilling (Virchow's *Archiv*, Bd. lxxxiii, S. 77) has described how epithelioma is propagated along the lymphatics and the nerves; and, after reading his paper, I carefully examined my sections with special reference to these two points, but could find nothing to lead me to suppose that in this case the lymphatics were specially involved, whilst the nerves seemed rather to resist the morbid action, for I found their transverse and oblique sections apparently quite normal, and yet entirely surrounded by the epithelial cells.

The mediastinal portion of the visceral pleura, which the tumour had also invaded, was examined; but the development of the growth in this locality so closely resembled that just described that the same drawing and description will answer equally well for both, with the sole unimportant exception that in the pleura, as also in the kidney and bone, a good many cells contained pigment granules, none of which I observed in that part of the tumour situated in the bronchial wall.

*Examination of the Bone.*—The sole distinction which I was able to observe between the characters of the growth in the rib and in the vertebræ, was that in the former it appeared to be more completely developed, and, as a consequence, the nests were more numerous. The manner of its origin and progress seemed to be identical in both localities, and it will therefore be unnecessary to describe them separately.

A section of the bone presents large irregular spaces of rounded outline, separated by narrow trabeculæ of perfectly normal structure (fig. 2, *a, a*). These spaces would seem to result from the erosion of the walls of the Haversian canals, partly because of their shape, and partly because the bone corpuscles are all arranged with their long axes parallel to the margins of the spaces. The centres of these cavities contain more or less closely aggregated masses of epithelial cells (fig. 2, *b, b*), which are, for the most part, ovoid in shape, and provided with a large nucleus and distinct nucleolus. They exhibit the

typical arrangement in "nests" in those places where the tumour is most completely developed.

A large number of the cells contain considerable accumulations of pigment granules, a point which, as above stated, was found to characterise the tumour in all its various situations except the bronchus.

In addition to the growth itself these spaces contain a large amount of the "moelle fibreuse" of Cornil and Ranvier (fig. 2, *c, c*); this consists of masses of closely packed, very long, spindle-shaped cells, each provided with an elongated nucleus. It is found in close proximity to the bouy trabeculæ, the fibres in many cases appearing to spread out from them in a kind of stream; usually the fibrous material lies alongside the trabecula in contact with it, but separated by a clear line of demarcation; but in some positions no such boundary can be traced, and the fibres appear to grow directly out from the bone (fig. 2, *d, d*). The bone corpuscles, too, can be traced passing from their lacunæ, when these are opened by the erosion of the bone, and taking part in its formation.

There is sometimes also a clear space between this tissue and the masses of epithelial elements, from all which it may be concluded that the former is really no part of the tumour, but merely the product of the inflammation set up by the presence of foreign elements.

The margins of the hollow spaces in the bone are, for the most part, of a smooth even curvature, but here and there this regularity is broken by small depressions of approximately semi-circular outline, and in many cases these are occupied by small cells with round nuclei, which stain deeply.

I have little doubt that these are concerned with the destruction of the bone, but I have hitherto failed in finding conclusive evidence of this.

*Examination of the Bronchial Mucous Membrane.*—Although the mucous glands appeared in those sections which showed the tumour advancing in the bronchial wall to be quite normal, I nevertheless found, in sections taken across some smaller bronchi, appearances indicating that morbid changes were taking place in them; although the alveoli were still of quite normal size, the epithelium had lost its columnar regularity, and the individual

cells had become swollen and rounded, so that the lumen of the alveolus was quite choked up with the mass of cells (fig. 3, *c*).

These cells were also proliferating, as might be seen in the alveoli represented in fig. 3, *a* and *b*. In *b* was a mass of protoplasm, in which were six separate nuclei, which had, moreover, become much more deeply stained than those around them, indicating, of course, that they were in a condition of more active vitality. In the alveolus (fig. 3, *a*) was seen near its centre a group of small cells, which appeared exactly as though they resulted from the breaking up of a similar multi-nucleated mass of protoplasm.

I could not find any glands in which the morbid process was further advanced than this, and I am therefore unable to confirm the statements of Langhans (Virchow's *Archiv*, Bd. liii, S. 470), that the alveoli increase in size, lose their *membrana propria*, and eventually break up into strings of cells, which grow out into the connective tissue in all directions.

*Examination of the Kidney.*—In this organ the first variation observed from the normal structure is a slight interstitial nephritis, an increase in the intertubular connective tissue (fig. 4, *b*), caused by the irritation of the approaching foreign growth, and completely parallel to the formation of "*moelle fibreuse*" which has been described in the bone.

The connective-tissue corpuscles increase gradually in number, and give rise to a small round-celled infiltration between the renal tubules; in the last stages, however, of the development of the tumour this interstitial tissue becomes less abundant, owing to the enormous increase in the epithelium.

Changes in this latter proceed concurrently with the processes just described. The cells, which at first appeared somewhat shrunken (fig. 4, *c*), owing no doubt to the pressure of the intertubular tissue, swell and become rounded, thus losing their columnar arrangement; their nuclei become more conspicuous, and at length they quite occlude the tubules (fig. 4, *d*), which in consequence do not stand out so clearly as in the normal kidney.

It is at about this stage that the connective tissue attains its maximum development; henceforward it begins to diminish in quantity.

The epithelial elements now increase rapidly in size, and form

in transverse sections large irregularly rounded groups (fig. 4, e), which are the representatives of the former tubules, all the intermediate stages between them having been observed. Eventually they give rise to the usual epithelial nests.

This account of the formation of the tumour in the kidney is essentially the same as is given of primary epithelioma in this organ by Waldeyer (as quoted in Birch-Hirschfeld's *Lehrbuch*).

*Conclusion.*—From the above I think we may look upon the lung tumour as primary, and the others as secondary. As regards the origin of the primary growth I can express no opinion; it may have arisen from the glandular epithelium or from the epithelium of the lung itself; in its further progress, however, it seemed to attack more especially the sub-glandular connective tissue. In the kidney its development from the glandular epithelium was clearly established.

The chief interest of the case lies in the nature of the epithelioma which belongs to the pavement-celled type, and its occurrence in these organs without any trace of it in the usual situations (external surface, junction of this and mucous surfaces lined with pavement epithelium) is a matter of the greatest rarity. I have been informed by Mr. Young that at the *post-mortem* examination the most careful search for any tumour, ulcer, or cicatrix in these localities gave only negative results.

The gradual change from a carcinoma with large alveolar spaces (corresponding more to the type known as scirrhus) to a tumour with the structure of an epithelioma occurs not unfrequently in such organs as the liver; but in this case the fully developed tumour, wherever found, was always of the epitheliomatous type (with nests), and nowhere resembled any other variety of cancer.

Before concluding I wish to record my thanks to Dr. Dreschfeld for having given me the materials for this investigation, as well as for the use of his laboratory and much valuable advice during its progress.

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## EXPLANATION OF PLATE XIX.

Fig. 1 ( $\times 200$ ). Longitudinal section through the bronchial wall. *a*, Margin of lung parenchyma with deposit of carbonaceous matter in the lymphatics; *b, b*, normal connective tissue; *c, c*, connective tissue invaded by the tumour; *d*, completely developed epithelioma; *e*, bronchial mucous glands; *f, f*, sections of cartilaginous rings; *g*, rounded infiltration between the alveoli.

Fig. 2 ( $\times 200$ ). Section of diseased vertebra. *a, a*, Trabeculae of bone; *b, b*, masses of epithelial cells; *c, c*, "*moelle fibreuse*"; *d, d*, places where this appears directly continuous with the bone; *e, e*, hemispherical hollows containing small round cells.

Fig. 3 ( $\times 550$ ). *a, b, c*, Separate alveoli of the bronchial mucous glands.

Fig. 4 ( $\times 200$ ). Transverse section of the kidney. *a*, Normal gland; *b*, increased intertubular connective tissue; *c, c*, shrunken tubular epithelium; *d, d*, tubular epithelium swollen and proliferating; *e*, large groups of epithelial cells developed from the tubules.

*N.B.*—Although figs. 1, 2, and 4 are shown as magnified 200 diameters, most of the details were drawn under a power of 550 diameters.

RESEARCHES INTO THE HISTOLOGY OF THE CENTRAL GREY SUBSTANCE OF THE SPINAL CORD AND MEDULLA OBLONGATA. By W. AINSLIE HOLLIS, M.D. Cantab., Brighton. (PLATE XX.)

THE spinal canal forms a convenient standpoint whence to consider the changes that take place in the grey columnar mass of the central nervous system as it passes upwards through the cord and medulla oblongata. It is around this tubular organ that the neuroglia of Virchow, the reticulum of Kölliker, or, as I prefer to call it, the *polio-synectic*<sup>1</sup> tissue is arranged. Throughout its length the canal, as is well known, is lined with columnar (ciliated) epithelium. In children and other young animals it is otherwise. I have observed, for instance, a relic of the developmental involution of the epiblast in a boy aged eight years, as far down as the lower cervical region, where a furrow separating the epithelial lining opposite the posterior fissure marked this embryonic peculiarity (fig. 1). The subepithelial tissue, which is indistinguishable from the synectic proper, and of which it forms a part, passes in such cases through the posterior fissure in the form of a triangular plug of membrane (as viewed in a transverse section), and completes the wall of the canal on that side. The triangular plug of membrane I have occasionally traced as filling the greater part of the posterior fissure as far down as the lower lumbar region in adults. It is, therefore, probable that development rarely eradicates all traces of the primitive involution of the ectoderm. In the medulla, the canal, after occupying a nearly central position at the lower part, gradually passes backwards as it ascends to the level of the calamus, where, as we know, it terminates in the fourth ventricle. In the newly-born kitten the quadrangular canal, on a level with the lower part of the olivary bodies, is lined on the three anterior sides only with columnar epithelium (fig. 2). The fourth and posterior wall is filled by the plug of synectic membrane above mentioned. At the

<sup>1</sup> Πολιός, grey; συνεκτικός, binding. For brevity, I elsewhere call this tissue "synectic."

calamus the epithelial lining invests the floor of the fourth ventricle.

Beneath the columnar layer the epithelial elements are observed in well-stained specimens to form more or less concentrically arranged layers around the canal. The deeper cells (as is usual) become more spheroidal in shape the farther they are removed from the lining membrane. The flattened nuclei of these cells stain deeply, especially if hæmatoxylin is the reagent, and when observed under a high power, such as a Zeiss's F, appear to be similar to the nuclei of the polio-synectic membrane elsewhere. In the young these nuclei are more numerous than in adult life, and they may be seen in the former case to thickly stud the developing nerve fibres of the white columns. In a longitudinal section of the cord of a kitten a week old I perceived these nuclei to be arranged by twos and threes in lines and in close approximation (fig. 3). Each group appeared to be surrounded by a tubular investing membrane, and from its situation and relationship to the adjacent tissues, I assume that the nuclei within it are deeply concerned in the differentiation and growth of the white nerve fibres. In adults each nucleus will be found embedded in a star-shaped cell of synectic tissue, whence fibres radiate in many directions (fig. 4). Deiters has already described these cells. As the nucleus stains far more readily than the rest of the cells, its peculiar formation is not always readily observable.

The similarity in the size and shape of the synectic tissue nuclei, and the close relationship which evidently exists between this grey tissue and the lining membrane of the canal as observed in the posterior commissure especially, and its numerously-radiating septa, would seem to support the idea that it is rather an outgrowth of the involved epiblast than derivable from the subpial membrane (or mesoblast) as Kölliker conjectures. It is possible that both methods of development may really take place in the formation of the inner and outer layers of the synectic tissue, the tissue framework of the central grey columns being specially of epiblastic origin, whilst the radiating septa of the circumference may be to some extent mesoblastic.<sup>1</sup>

<sup>1</sup> Before finally dismissing this part of the histology of the cord and medulla, I may draw attention to the fact that certain pathological states seem to influence

The much greater proportional size of the central grey columns to the white in young animals, together with the important rôle played by the synectic tissue nuclei as above mentioned, leads me to infer that during growth white medullary fibres are gradually evolved from the peripheral layers of the central grey cellular material.

1. *The Ganglion Nerve Cells.*—Besides the nuclei and cells above described, which are a part of the synectic tissue of the cord and medulla, there are various cellular elements which belong to the nervous tissue imbedded within it. These merit a short description, as my observations differ somewhat from those of preceding histologists. I think they may be classified under three distinct heads, according to certain peculiarities of shape; their variable size appears to be of less anatomical importance.

In the anterior cornua of the cord, and their extensions to the medulla, and in the hypoglossal and other so-called nerve nuclei of the latter body, we find (as is well known) the giant ganglion cells of histologists. These cells, although extremely variable in shape, have certain well-known peculiarities which mark their species. Their bodies are usually more or less fusiform, with many processes extending from them, and they invariably include a large well-developed nucleus. Histologists usually consider that one of these processes becomes a medullated nerve fibre, whilst the others divide dichotomously, and ultimately form a delicate nervous plexus. I cannot say that I have been able to see this peculiar process arrangement satisfactorily. I believe that in some instances two or more nerve fibrillæ may originate in one ganglion cell, and as regards the ultimate distribution of the branched processes I am ignorant. I have, however, seen two ganglion cells occasionally to be united by a fibre (fig. 5). In adults the nuclei of these cells stain readily with the usual reagents, the cell plasma not quite so easily. This circumstance has led some pathologists to suppose erroneously that such an appearance indicates a morbid change in the cell plasma, but this is not

the development of the synectic tissue nuclei. In a transverse section of the cord through the roots of an upper cervical nerve (from a man who died of traumatic tetanus after an injury to the hand) I found the posterior roots with the *substantia gelatinosa* of Rolando infiltrated by large numbers of these bodies, which were far more numerous there than usual in adults.



necessarily the case.<sup>1</sup> When highly magnified, the nuclei of these cells have a roughly granular appearance (figs. 5 and 6), a condition which might imply an agglomeration of convoluted fibrillar contents, the result of previous karyokinesis. In infants and young kittens these large ganglion cells occasionally have two nuclei (fig. 8), and the polar processes are frequently absent, or, when present, are comparatively short (*cf.* figs. 6 and 7). It seems that polar prolongations are developed in many instances with the growth of the animal. Some one or two from each cell serve ultimately for the conveyance of nerve-vibrations, and the others become split up by dichotomous ramifications until their extremities are lost in the surrounding synectic tissue. The latter prolongations may be subservient to the nutrition of the cell. I can scarcely imagine that they play an important part in the conduction of nervous impressions. I believe, with Golgi, that all the processes attached to a cell may be branched, but I cannot assume with him that some cells form nerve reticulations and others do not, as I have been unable to trace the former arrangement in any sections under my observation.

2. *The Pyriform (unipolar ?) Cells.*—The vesicular columns of Clarke, and the tractus intermedio-lateralis in the spinal cord, and the layer of grey matter which separates the olivary nucleus on its inner and posterior aspects from the rest of the medulla, afford in their cellular contents numerous examples of the second form of giant nerve ganglion, the pyriform or ovoid cell. These cells, which may be occasionally observed in other parts of the cord and medulla, differ in so many important respects from the multipolar cells above mentioned that I have ventured to give them a separate description. Their average size is smaller than that of the multipolar cells, their bodies stain more readily throughout than do the others, and we consequently find their nuclei somewhat less prominently visible in prepared sections. In certain parts of the cord they are found closely congregated in cell nests.<sup>2</sup> The cell processes are limited to one (or it may be two prolongations in some cases) to each ganglion. In the

<sup>1</sup> Hæmatoxylin, carmine (ammoniacal) and picrocarmine solutions are the reagents I have generally used for staining nerve tissues.

<sup>2</sup> Especially is this the case in the filum.

tractus intermedio-lateralis, the solitary cell process is seen frequently to pursue an outward lateral course with other nerve fibres running from the posterior outer angles of the anterior cornua. Among these cells the process-root (as it issues from the cell-body) is generally transversely striated. Under a Zeiss's F, with Abbe's condenser, this striation assumes the appearance of a spiral fibre wound around a direct one, or of several flattened discs placed edgeways against each other (figs. 9 and 10). The researches of Beale and Arnold have proved the existence of somewhat similar cells in the frog's sympathetic, and S. Mayer has noticed spiral fibres in the sympathetic cells of the higher vertebrata. The spiral (if such it be) and the straight fibre around which it is coiled, I have traced to a polar plasmic agglomeration in the cell body.

3. *The Cells of the Olivary Nucleus.*—The cells included within the corpus dentatum of the olivary nucleus differ in many respects from either of the preceding. Their size, which varies less than that of the other cells, is considerably smaller than the average of either of them. In shape they are spheroidal, and so far more nearly approach the pyriform than the multipolar ganglion. They are usually unipolar, and have a delicate connecting stroma of fibrillæ between their bodies and the adjacent synectic tissue. I have observed these cells to be connected to each other occasionally by short processes (fig. 11). In the figure depicted in the accompanying plate there are no fewer than three cells so united. This mode of union is by no means common, although a somewhat similar arrangement has been described by S. Mayer as occurring in the sympathetic system.

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#### EXPLANATION OF PLATE XX.

1. Transverse section of central canal and adjacent grey matter of spinal cord, from a boy aged eight, showing the fissure in the posterior epithelial walls at *a*; *b*, wedge-shaped prolongation of grey matter from posterior commissure; *cc*, epithelial lining membrane of canal.

2. Transverse section of the medullary canal of a young kitten, a few days old, on a level with lower edges of olivary bodies. The anterior (ventral) aspect *A*, and the two lateral borders *c c* of the

canal are lined with columnar epithelium; the posterior border *P* is occluded only by a wedge of grey synectic tissue, *syn*.

3. Synectic tissue nuclei in a line, showing the probable mode of formation of the white medullary fibres. From a longitudinal section of the dorsal cord of a kitten one week old. Highly magnified.

4. Two synectic tissue corpuscles from the medulla of a man.

5. Two multipolar ganglion cells from human medulla, showing their union by a dichotomous fibre at *a*.

6. Group of multipolar cells from hypoglossal nucleus near the calamus. Infant eighteen months.

7. Group of similarly situated cells from a boy, aged nine years. (This patient died of hydrophobia.)

8. Giant cell with double nucleus, from the spinal cord of a kitten, five days old. (Müller's fluid and picrocarmine solution.)

9. Pyriform cell with a spiral (?) terminal fibre surrounding a direct one from the anterior cornu at the junction of the medulla with the cord. Longitudinal section. Human adult.

10. Pyriform cell from the vesicular column of Clarke, in the upper dorsal region of a boy, aged eight years; *f*, nerve prolongation with transverse striation (spiral fibre?). Highly magnified.

11. Three olivary cells in juxtaposition with short communicating fibres. Human adult.

ON THE MEMBRANA TYMPANI. By JOHN M. CROMBIE,  
M.A., M.B., &c.

SINCE the appearance of Helmholtz's treatise on the mechanism of the ossicles and membrane, translated by Hinton, there seems to have been no important attempt at their further investigation. This can hardly be attributed to his treatise having furnished a satisfactory solution of their mode of action. An explanation of the mechanism of the *membrana tympani* that takes no account of the *membrana flaccida*, its upper portion, can at best, even if otherwise correct, be but incomplete and defective. Helmholtz asserts that the lower firm portion of this membrane is the only part concerned with sound waves, and it is perhaps for this reason that he gives it exclusive attention. But whether the upper portion has any share with the lower in sound reception or not, it certainly has a purpose relating to sound, and an influence on the function of the lower which renders its consideration indispensable. Such bisection, indeed, excludes one-half of the machinery of the membrane. In other respects the treatise is hardly more satisfactory, which is to be regretted, as it is the most complete of its kind, and many of its conclusions have obtained a wide currency in books of physiology as established principles of unquestionable truth. It seems to have failed as a whole, because an interpretation is sought on the principles of acoustics, although Helmholtz has succeeded in some degree in their application, yet even he felt how different the *membrana tympani* is from the tense membranes known to acoustics. "It differs," he says, "from those membranes that have hitherto been investigated and employed in acoustics in this respect that it is curved."<sup>1</sup> This simply means that a completely satisfactory explanation of the mechanism of the ear cannot therefore be looked for, since the *membrana tympani* is quite peculiar in this important particular. "Hitherto," says he, "the acoustic properties of such curved membranes have not been practically investigated: perhaps the most that can be

<sup>1</sup> *New Syd. Soc.*, vol. lxii. p. 141.

said is, that I have seen use made of a curved piece of leather as a sounding-board on an Arabic percussion instrument which was played on in the Tunis café in the last Exposition in Paris.”<sup>1</sup> It is obvious that this does not amount to much; and, in endeavouring to apply the principles of a science as yet so admittedly inapplicable, it seems Helmholtz, in spite of the mathematical ability displayed in his work, has not attained any very valuable result. It is true a number of conclusions have been arrived at, but they relate for the most part rather to aural curiosities than matters of broad and important interest. The position of the membrane most favourable for accurate hearing is not indicated, but the cause of certain rattling sounds, “such as one hears in a musical instrument when something is loose, or in a tuning-fork which has not been fastened quite firmly to a sounding-board,” is reduced to a physiological explanation of much nicety. The adaptation of the membrane to tones of different pitch is not once alluded to, but there is elucidation “for the peculiar sensation of buzzing in the ear, which is felt in the combination tones of two powerful soprano voices in a passage of thirds.”<sup>2</sup> It is those interpreters—the acoustic principles, not the anatomy of the ear—that lead up to these results; but whether they are correct or not, one might be permitted to question whether the author is not deceiving himself, when, also on acoustic grounds, he declares that in the midst of other sounds he can detect as a distinct sensation the rattling of the cogs of the malleo-incudal articulation, “as a rumble in separate shocks.” My object in referring to these things is not to disturb the belief of those who have faith in whatever authorities write, but to insist on the important fact to aurists, that they must not look too implicitly to acoustics—at any rate, in its present state—for a useful and trustworthy account of what lies at the foundation of otology—the mechanism of the membrana tympani. The organ can be looked at and examined in its most delicate and minute points to see what it is, to better purpose than to see what it should be on any theory of acoustics. And such a work as this of Helmholtz’s is rather a stumbling-block than a guide, since it fosters the belief with its mass of mathematical formulæ that, in regard to the investi-

<sup>1</sup> *New Syst. Soc.*, vol. lxii. p. 146.

<sup>2</sup> *Ibid.*, pp. 137, 138.

<sup>3</sup> *Ibid.*, p. 138.

gation of the ear, this is the proper mode of approach. It is not to be prejudiced against mathematics, here or elsewhere, to accept no formulæ as expressions exact or approximate of truth, till one has convinced himself that the elements manipulated are the genuine factors involved in the problem. How this can possibly be the case with Helmholtz's elements, a review of some of the leading points of his interpretation of the mechanism of the membrane will show, and this is entered on, not simply for the purpose of disproving the opinions of this particular treatise, but of professional opinion on the subject generally, of which it is the best and most authoritative exponent.

It is proper to state that Helmholtz himself confesses to some of the shortcomings of his treatise, as, for example, that he has given no consideration to the facts that the meridian arcs of the membrane are connected together; that the distances between them increase towards the attached edge of the membrane; that they are bound together by circular fibres, and cannot move without stretching them, &c., &c.<sup>1</sup> But his conclusions have nevertheless been admitted into physiology without any deductions on those grounds, or on the more important ground, of which he takes no notice, of the entire omission of the function of the *membrana flaccida*. He undertook the investigation, he explains, chiefly because he could not reconcile the idea, held by certain writers, of a very yielding joint, supported almost entirely by a weak and relaxed covering membrane, such as that of the malleo-incudal articulation, serving as a connection in the middle of a firm lever. But his own examination of this articulation, if correct, establishes that, furnished with the cogs already referred to, it is not the relatively weak joint he took it to be. Regarding the general nature and action of this membrane, he adopts the received opinions, so that, in the main, he has nothing new to advance. Thus, to begin with, he lays down that the drum is a tense membrane, and that, as occasion requires, it is rendered more tense by being bodily drawn inwards along with the whole manubrium, through the action of the tensor tympani muscle. It is in endeavouring to demonstrate in detail how such movement is effected by the various agents concerned that

<sup>1</sup> *Nerv. Syst. Soc.*, p. 145.

he puts forward his own views. And, apart from the merit of his discovery of the interlocking teeth of the malleus and incus, about the existence of which, I suppose, there is no doubt, they are by no means to be preferred to those of previous expositors, who are distinctly more clear, and far less inconsistent. Thus, it was held by Weber that the malleus and incus form together a firm angular lever, whose axis of rotation runs from the point of attachment of the processus longus of the malleus to the top of the processus brevis of the incus. That, taking into account the outward inclination of the membrane in its antero-posterior attachment, gives an axis running through the malleus and incus in a straight line between the points indicated, around which the entire apparatus of ligaments and muscles naturally resolve themselves for the only possible movement of the malleus, and with it the membrana. Weber did not indicate that movement, as it is impossible he should, looking, as he did, only from the tympanic side of the drum; but his axis of rotation is undoubtedly correct, as the arrangements above alluded to prove. Helmholtz, on the contrary, adopts an axis of rotation passing in a line running between the spinæ tympanici major and minor, as he names the two osseous projections at the anterior and posterior corners of the inner end of the upper wall of the meatus. This axis accordingly passes partly through the processus gracilis, partly through the membrane, and partly along a ligament at an acute angle behind the membrane. In order to reconcile the movement that would result from such an axis, with the more obvious necessities of the movement of the membrane, a number of subsidiary movements have to be introduced, which are not in keeping with the anatomical machinery involved, and which are so intricate and ill-defined as to make an exact calculation of their resultant impossible. For instance, if the malleus were strictly to revolve round its axis band, thus defined as an axis of rotation, it is admitted that the umbo would not move in a direction normal to the plane of insertion of the membrane—for the purposes of audition a fatal deviation. To correct this, two slight alterations in its movement, through its connection with the incus, are discovered for the malleus. “(First) As the axis band of the malleus is directed obliquely to the plane of insertion of the

membrane, each inward movement of the malleus handle would cause also a backward movement of the umbo. But, at the same time, the malleus head would be drawn backwards by the incus, and so the handle receive an opposing motion forwards. (Secondly) As the umbo of the membrane lies further from its plane of insertion than the axis of the malleus (excepting at most its foremost end at the *spina tympanici*), each inward motion of the malleus handle will cause a small upward displacement of the umbo, *i.e.*, in a direction towards the head of the malleus. This motion, and that which we have just explained, namely, that the malleus, as a whole, is drawn somewhat downwards by the incus, act in opposite directions. Thus, both deviations in the movements of the umbo are compensated, and thus there remain only the movements that take place perpendicular to the plane of insertion of the membrane."<sup>1</sup> It would require too much space to enter on a criticism here of how the incus thus comes to the rescue of the malleus, with equivalents of force exactly equal and opposite in direction. It is a very recondite, and not very lucid, exposition in the hands of Helmholtz himself, based on the relationship of certain ligaments—but it does not aim at being a quantitative mechanical demonstration, but only a general indication of the direction of the forces, the exactness of the quantities being taken for granted. A general objection to it arises from the fact that the anatomy on which it relies does not coincide with that recognised by the best authorities. The *spina tympanici minor* is not even alluded to by Henle. But, even looked at mechanically, the theory is inconsistent with itself, since the axis of rotation thus modified is really no longer in the line of the *spinæ* assigned to it, but approaches, in fact, as nearly as possible that already mentioned as pointed out by Weber.

Not only, however, is the treatise retrograde in this very important particular, but, by starting with the accepted notions concerning the action of the tensor tympani, and the condition of the drum as a permanently stretched membrane, it is marred by very serious errors.

What proof is there that the drum is maintained normally in

<sup>1</sup> *New Syd. Soc.*, vol. lxii. p. 129.



a state of tension? Let it be remembered that tension in elastic or inelastic bodies implies the expenditure of force, and that tension is only maintained while force is in action. If the membrana tympani is permanently tense, it must be from some permanently extending force. Helmholtz says :—" Its tension is conditioned by the handle of the malleus, which draws it inwards, and which is itself maintained in position by its ligaments and by the elasticity of the tensor tympani."<sup>1</sup> From this account it might appear that the resistance to this active strain of the membrane, its ligaments and muscle fell to be borne by the bone surrounding the membrane and affording it attachment. If the membrane is robust, so also is the bone, and perhaps such a struggle might be compatible with their mutual constitution ; but if so, it is an unique instance in the human body of ceaseless antagonism and consumption of energy in maintaining a simple *status quo*. There is opposition between anatomical elements elsewhere, but without exception they are allowed intervals of repose, and breathing time to recuperate, and their function is movement, not balanced and motionless equilibrium. Here we have a picture of uninterrupted strain, involving a number of most delicate agents, addressed to the maintenance of a simple dead weight. "Peu de moyens, beaucoup d'effet"—Nature's universal principle is here beheld reversed. But, according to Helmholtz, it is not on the sulcus tympanicus that the whole pull of resistance falls—there is a perpetual struggle in the midst of the membrane itself, between two of its constituent sets of fibres. "Were the radial fibres of the membrane alone in existence, and not joined with transverse fibres, they would extend in straight lines (from the malleus handle to the margin of attachment). In fact, they do not do this, but assume a curved form, convex on the side of the meatus, whence it is to be inferred that they are drawn to each other by the ring fibres, and that these latter are also put on the stretch. In fact, in the quiescent membrane there is no other force that can maintain the radial fibres in their curved form besides the tension of the ring fibres." Quiescent is not a happy term for a membrane which is thus the seat at all times of internal contention, and the centre besides of application of external opposing

<sup>1</sup> *Ibid.*, p. 141.

forces—but, *sic venio verbo*, it is quiescent comparatively to the time when, according to accepted opinion, the tympanic muscle puts forth its further tensating energy. It must be objected, however, to this life-long and incessant contest between the radial and circular fibres, that it implies the possession of a property by the latter which they are known to be entirely destitute of, and which Professor Helmholtz only presumes must exist, in the face of facts, for the purpose of this tension theory. If the two sets of fibres were of the same character, and a force applied which acted equally on both, such as that assumed in connection with the manubrium, then their distension would simply be equal, and the same relative distance between the radial fibres would still be maintained. They would inevitably extend in straight lines, and no curving would take place. But, if we assume that the circular fibres are strongly elastic, and that it is this property that is called into play when they are stretched, then they will tend to shrink back to their previous dimensions, approximating the radial fibres, and affording, perhaps, the desired curvature. Unfortunately, the circular fibres are possessed of no such property, and Helmholtz does not in the least dispute it as a matter of fact. Thus, speaking of the fibrous skin constituted by these two sets of fibres, and which forms the middle layer of the membrane, he says :—"The fibres of this layer are very firmly stretched bands lying close to one another, and oppose a great resistance to any extension. They are distinguished by their great inelastic resistance."<sup>1</sup> Henle says that, in regard to this central layer, the important point to be noticed is that its fibres, though in chemical character resembling ligamentous tissue, are distinguished from common ligamentous tissue in these particulars, that they are neither spiral nor fibrillated, and that the interspaces in the web they form contains no elastic tissue, while there are present only minute nuclei, and these very sparsely distributed.<sup>2</sup> The force of this description, which accords with that of the best authorities, is, that the fibrous skin is wholly destitute of elastic properties; it is provided with no special elastic tissue, and its own fibres are perfectly straight, without even the slight tinge of extensibility that would belong to them had they been

<sup>1</sup> *Ibid.*, p. 114.

<sup>2</sup> *Anatomie Des Menschen*, Band ii. S. 735.

of the spiral or fibrillar form. The finishing stroke is given to their rigidity by all but the absence of even rudimentary nuclei. Yet it is this non-existent force which Helmholtz evokes to hold the radial fibres in their attitude of curvature. "There is no other force that can maintain the radial fibres in their curved form besides the tension of the ring fibres."<sup>1</sup> This attribute, however, of the tympanic membrane, is a cardinal one, and, if the theory of tension cannot explain it consistently with facts the idea of a permanently stretched membrane must be abandoned. It involves, besides the departure from the great precedents already alluded to, a contradiction to all experience of similar structures elsewhere, for no membrane known to us in the physical world could endure perpetual strain without elongation or rupture. It is not consistent with clinical experience, for the membrane, when cut into, does not gape as a stretched membrane would do, but remains with the straight cut edges in apposition. So says Helmholtz himself. "It is to be considered not as an elastic and yielding membrane, but as an almost inextensible one; its very slight extensibility is seen if, either in its natural situation or when removed, it is spread on a glass plate and torn with pins." There is nothing absolute in nature, but the drum of the ear approaches as nearly as possible to an inextensible membrane as anything could. Yet Helmholtz admits that in his investigation he takes no account of the fact "that the curved natural form of the membrane cannot exist without every force which presses the malleus handle inwards, also stretching and expanding the circular fibres. On account of the irregularly proportioned form of the membrane, no full analysis of the mechanical actions of these relations can be given. For that purpose the tension and the grade of the elasticity of the circular fibres must first be known."<sup>2</sup> We have already seen that there is no such elasticity. Professor Helmholtz would be able to give, he says, a mathematical formula somewhat better corresponding to real proportions, if he were permitted to imagine, instead of the real, an ideal membrane, and among other variations required is a strong stipulation in regard to these same circular fibres. "The radial fibres which should traverse the meridians of

<sup>1</sup> *Loc. cit.*<sup>2</sup> *Ibid.*, p. 145.

such a surface, might be considered as inextensible, but the circular fibres must have a certain degree of elasticity in order to remain stretched." <sup>1</sup> But what really is the use of a mechanical analysis, or a mathematical formula for physiological ends, that is founded on an imaginary anatomy at cross purposes with real anatomy. Nature has made the circular and radial fibres of this layer of the membrane of identically the same material, that is, of the most inelastic substance to be found among tissues, and however elegant the formula which takes for one of its elements the contrary, it is incorrect and fallacious. Further, an incorrect physiology leads to mischievous modes of treatment, medical and surgical, and in relation to the action of the tensor tympani in further stretching this hypothetically stretched membrane, there are wrong ideas entertained, which do result in very erroneous treatment. Surgeons, in these enterprising days, are not deterred by any difficulty in planning operations, if they are encouraged by any feasible probability of obtaining the result desired, and aural surgery is not so fertile in its openings for operations that any avenue presenting one should be neglected. It consequently follows that the tensor tympani, situated though it is in the most inaccessible of regions, is nevertheless assailed by the irresistible tenotomist, who believes that his patient is suffering from over-stretching of the membrane and its consequences, owing to spasmodic action of its muscle. This physiology is of a piece with that we have been examining, and a careful consideration of the demonstration given by Helmholtz on the subject, the fullest yet advanced, will show that it breaks down in the same manner, since it has to fall back on the play of imaginary forces. In examining, therefore, the action of the tensor tympani, we are pursuing the same subject from another point of view, in which the tension theory will be seen to be even less tenable.

We must first take account of the numerous structures that are said to be put on extra tension by the contraction of the tympanic muscle. "Primarily it draws the malleus handle inwards, and with it the membrane, but, at the same time, it acts on the axis-band of the malleus, which it draws inwards

<sup>1</sup> *Ibid.*, p. 145.

and stretches. Thereon the head of the malleus is moved away from the incudo-tympanic articulation, and the ligaments of the incus are stretched, as well those towards the malleus as those at the point of its short process, which last is gained from the bone."<sup>1</sup> In short, with the single exception of the ligamentum mallei superius, not only the membrane and all its ligaments, but all those ligaments which fix the ossicles in their position, are said to be rendered tense by the action of this muscle. Now, although these structures are small or minute, yet, since they are all more or less inextensile, their stretching must demand a relatively large amount of force, since the force necessary to maintain such textures at the same degree of tension does not decrease equally to their size. A muscle with a long tendon does not require much greater expenditure of energy to put its fibres on the stretch than a muscle with a short tendon. If such fibres were highly elastic it would be different. We must not, therefore, be misled by the smallness of the parts concerned, since it is the degree of tension to which they are put that will mainly determine the amount of power necessary. Helmholtz<sup>2</sup> says:—"This ligament—the axis-band of the malleus—is always somewhat tense, even if the tendon of the tensor tympani is severed; but so long as this muscle exerts its strain transversely upon the axis-band, the tension of the latter is kept at a high point." What is true of the axis-band must be true of the membrane and other ligaments involved, true, that is to say, on the score of argument. Now, in order to qualify the tensor tympani for the exercise of an equivalently high strain, Helmholtz supplements its power fictitiously in two ways—first, by over-estimating its mechanical strength; and, secondly, by attaching a significance to its mode of action inconsistent with mechanical principles.

As regards the muscle itself, a more inadequate instrument, looked at anatomically, for such high pressure work could hardly be conceived. It is, as we know, about half an inch in length, and hardly a millimeter in section, consisting of fibres that run parallel to each other, and that end in a round and very slender tendon. Such a muscle, if placed in the most favourable circumstances, would seem a very pigmy in view of the gigantic

<sup>1</sup> *Ibid.*, p. 129.

<sup>2</sup> *Ibid.*, p. 122.

duty imposed on it; but when we consider, at the same time, that it is encased as in a sheath by the incompressible walls of a bony canal, and that its tendon acts on the malleus at a right angle to the muscular fibres, what are we to think? The first circumstance forbids all but the feeblest contraction, since it admits of only the least possible shortening of the fibres, or swelling up of the muscle; and the next circumstance reduces the effective force of even this attenuated energy by placing its operation at a mechanical disadvantage. Helmholtz omits all mention of these circumstances, but dwells on the occult properties of the muscle instead. "Since the tensor tympani is, on account of its feathered structure, equivalent mechanically to a muscle of much greater section and less length of fibre, we must regard its elastic strain, even without any active contraction, as a considerable force."<sup>1</sup> I cannot find the tensor described elsewhere by independent observers as a feathered muscle. Professor Henle, the most minute and exact of anatomists, in his description says:—"It passes over the septum tubæ into the tensoris tympani canal, which it traverses from one end to the other in bundles of fibres, that proceed parallel to the axis of the canal."<sup>2</sup> In Quain's *Anatomy* it is described as consisting of a long, tapering, fleshy part, and a slender tendon. Here, then, at least, is a hypothetical attribute; but be the effect of that as it may, how is it possible to reckon the elastic strain of such a muscle, apart from all contraction, as a considerable force? The elastic strain of the gluteus maximus may be somewhat, but only the merest fraction, of the full force of the muscle in active contraction, but the elastic strain of the tensor tympani must be, even on the same scale, as near as possible to a cipher. We are to judge of the potency of a muscle by its tendon, which will always be made stronger than the greatest muscular strain exercised through the muscle. Nature will not employ a rope unequal to the power applied behind it, but will certainly have it too strong rather than too weak. The tendon of the tensor tympani is a mere thread, and in its rounded shape and application to a single point, answers to the character of a muscle intended to act on a minute joint of easy flexibility, rather than a stretcher of such tense ligaments and

<sup>1</sup> *Ibid.*, p. 122.<sup>2</sup> *Anatomie des Menschen*, Band ii. S. 747.

membranes. Indeed, one must shut their eyes to the plain and simple facts of its anatomy and relationship, and get very much possessed in favour of secret and unsearchable properties to connect this tiny tendon with extensive effects on all the inextensible tissues surrounding it. "In reference to this," says Helmholtz, "the construction of the ear is very remarkable. By the traction of this one elastic mass of fibres (the tensor tympani), whose tension, besides, is variable, and can be adapted to the requirements of the case, in addition to its effect on the membrane and its ligaments, all the inelastic ligaments of the ossicles can simultaneously be placed in active tension."<sup>1</sup>

I think it is plain that Helmholtz, in overlooking the size and situation of the tensor tympani, and the mechanical disadvantage of its exertion on the malleus, and in seeking for attributes of force in undemonstrated structure and inconceivable elastic strain, summons up a force which does not exist.

It is obvious that if the membrana tympani, as an inextensible structure, is already in a state of considerable tension, a new force that shall raise it to a higher degree must not only balance the original tension, but exceed it by the amount of the tension to be superadded. Accordingly, the traction of the tensor tympani on the membrane cannot effect any change on its tension until it passes the point where it balances that tension. In all degrees below that it can only produce pressure on the spot where it is applied. Helmholtz's mode of mechanical action, as set forth in the action of the tensor, is the reverse of this.<sup>2</sup> Not only may a slight traction on a tense membrane slightly increase its tension, but, positively, a slight traction on such a membrane may very considerably increase its tension, if, that is to say, the traction is applied transversely. His words are:—"A very moderate tension of the tendon gives a very tense poise to the malleus. We must here consider that a slight tension which is exerted transversely upon a tense inextensible band is able to increase the tension of that band very considerably." This is erroneous. There is no charm in transverse application whereby a small force may become a large, nor whereby a force can add one tittle to its effect in this kind of tension production. It is only when the space through which the

<sup>1</sup> *Ibid.*, p. 180.

<sup>2</sup> *Ibid.*, p. 122.

power acts is proportionately increased that an enhanced action from transverse application comes into play, that is to say, the power multiplied into the space gives the effective force. As the space is increased the power may be proportionately diminished—the effective force remaining the same; but this condition is entirely excluded in the above case, because the space through which the tensor moves its tendon is infinitesimal.

Unless, however, the tensor were endowed with some such latent energies as above referred to, and liberated from the stern exigencies of mechanical law, it could never operate within its bone-bound walls, and with its angularly directed tendon, with sufficient force to augment the tension of the membrane and its appendages. But such additional might, even if wrought by magic, would not suffice by itself. Something more is required for the proper tension of the membrane than the action of the tensor muscle. Every increment of its power must be accompanied by an equivalent increment of the elasticity of the circular fibres, otherwise the membrane on being drawn inwards would lose its relative curvature. Again, then, must we draw *pari passu* with the fictitious development of the tensor's action on another fiction, the elasticity of the ring fibres.

The tension theory, indeed, is inconsistent with itself on every hand. How is it possible, if the membrane is to be regarded as an inextensible one, and already on the stretch, that it should move inwards when the tensor acts on the malleus? An inextensible membrane or band is not on the stretch if it is capable of further movement. Either, therefore, it is not already on the stretch normally, or, if on the stretch, then this inward movement is an impossibility. Anatomists have always had the greatest difficulty in reconciling any inward movement of the membrane with the preservation of their proper relation by the ossicles of the tympanum. There is a distance of only from two to three mm. between the umbo in the lateral wall and the promontorium in the median wall of the tympanum, consequently the approximation of even one millimeter would bring the lower end of the manubrium in contact with the crura of the stapes, which project a millimeter and a half horizontally outwards from the median wall. Again, in proportion to the inward movement of the membrane would the ossicula be forced inwards, and the



base of the stapes driven into the vestibule through the foramen ovale. It is manifest, however, from the form of the articular surfaces of the ossicles, and from their closeness together, as well as from the fact that they are tied up tightly to the walls of the cavity, that, if any, only the least possible inward movement is possible. Indeed, all the supremely delicate arrangements of the tympanum, down to such microscopic minutiae as the interlocking teeth discovered by Helmholtz, point to special provision against all but the merest vestige of movement inwards.

What would such movement accomplish? It cannot be associated in the least appreciable manner with any distinct function of the tympanic membrane, or with any purpose of audition. Indeed, the tightening and traction inwards of the membrana tympani by the so-called tensor muscle is a fiction without any assignable significance in physiology, while it is discountenanced by the facts of anatomy and the principles of mechanics.

*December 1882.*

AN ACCOUNT OF AN OBTURATOR HERNIA. BY W. S.  
RICHMOND, *St. Bartholomew's Hospital.*

IN the dissection of a female subject, a hernia was discovered passing through the canal for the obturator vessels and nerve in the obturator foramen on the right side. Although no constriction of the bowel had occurred, yet a portion of intestine had passed through this canal to the extent of an inch and a half, pushing before it two layers of peritoneum. After the removal of the intestine the parietal layer still remained *in situ*, forming a *cul-de-sac* large enough to admit the finger. In passing through the foramen it was contained in the oblique canal, formed by the horizontal ramus of the pubes above, and the arched border of the obturator fascia below, together with the vessels and nerve. Outside the pelvis, in its short extent, it followed the anterior division of the obturator nerve, having the pectineus and adductor longus in front, and the adductor brevis behind.

The distribution of the arteries was abnormal. The place of the obturator artery was supplied by a branch from the femoral, which was given off just below Poupart's ligament. It proceeded backwards over the horizontal ramus of the pubes, beneath the external iliac vein, curving downwards to pass through the canal above, and externally to the hernial sac. This differs from the usual course of the obturator artery when given off from the external iliac. It is generally found to proceed over the vein, not, as in this case, beneath it. This artery also gave off a pubic branch, which anastomosed with the deep epigastric. An artery, corresponding to the normal obturator, was given off from the anterior trunk of the internal iliac, and proceeded forwards in the broad ligament and along the brim of the pelvis, where it terminated by anastomosing with a few offsets from the artery previously described. Hernia occurred on both sides, but that on the left was insignificant. An incomplete inguinal hernia had occurred on the left side, but was arrested probably on account of the extreme constriction of the external abdominal ring.

It need scarcely be remarked that herniæ through the obturator foramen are exceedingly rare.

FIBROUS BODY ATTACHED TO THE HYDATID OF MORGAGNI. BY W. S. RICHMOND, *St. Bartholomew's Hospital.*

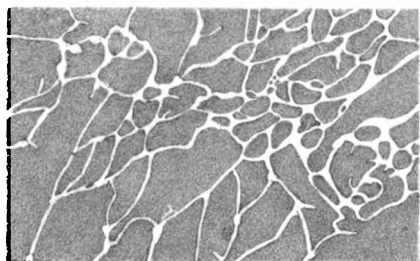
ADHERENT to the hydatid of Morgagni of either testis is a small fibrous nodule attached to its middle and under surface; it is white and glistening, hard to the touch, and firmly fixed. Some of its fibres can be traced to the root of the corpus Morgagni, and this seems to suggest that it is the remains of some foetal structure. Is it, then, the remains of another portion of Müller's duct? I think not; for if it were so, it would not be attached, as it is, to the hydatid of Morgagni, which is itself a remains of Müller's duct. It would form another separate hydatid, having a distinct attachment. These separate hydatids have been noticed by Dr. Banks. In his essay "On the Wolffian Bodies" he says: "I have seen two or three hydatids running from Morgagni's one, which I do not doubt were the remains of the duct" (Müller's); but these were "*running in a line from Morgagni's one,*" as one would naturally suppose. If the duct became obliterated at places, each part thus separated would have no connection with another part, and must have therefore a distinct attachment, unless it entirely disappears. This body, then, can hardly be regarded as another remains of the Müllerian duct; but may it not be the remains of a portion of the Wolffian body—that portion which generally disappears without undergoing any differentiation, viz., that part which lies between Müller's duct and the upper body of the testis? For if this portion is to remain at all, its remains must be attached either to the hydatid above or to the testicle below. In this instance it is attached to both. I think, therefore, that this nodule is the remains of a portion of the Wolffian body.

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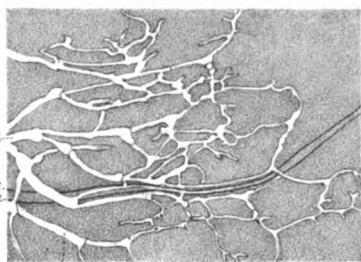
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*Fig. 1.*



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*Fig. 2.*



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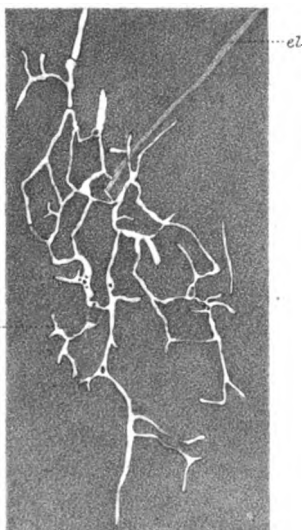
*Fig. 3.*



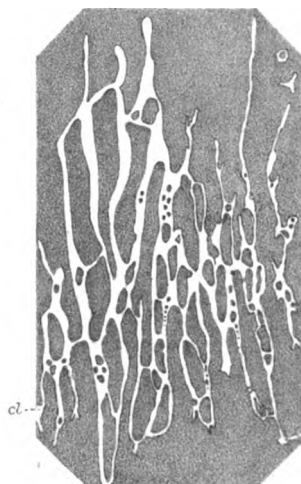
*Fig. 4.*



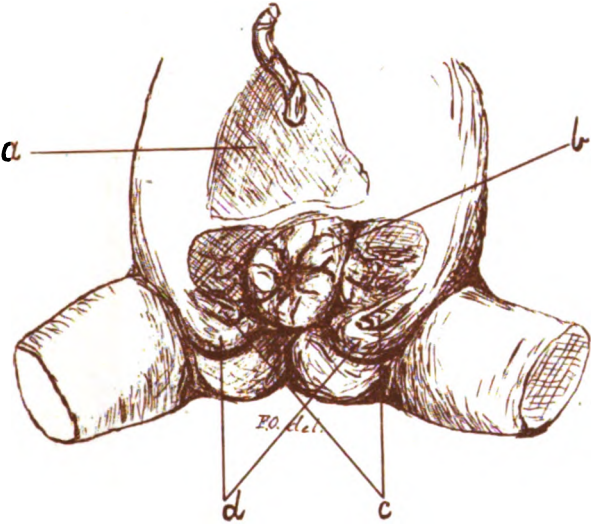
*Fig. 5.*



*Fig. 6.*



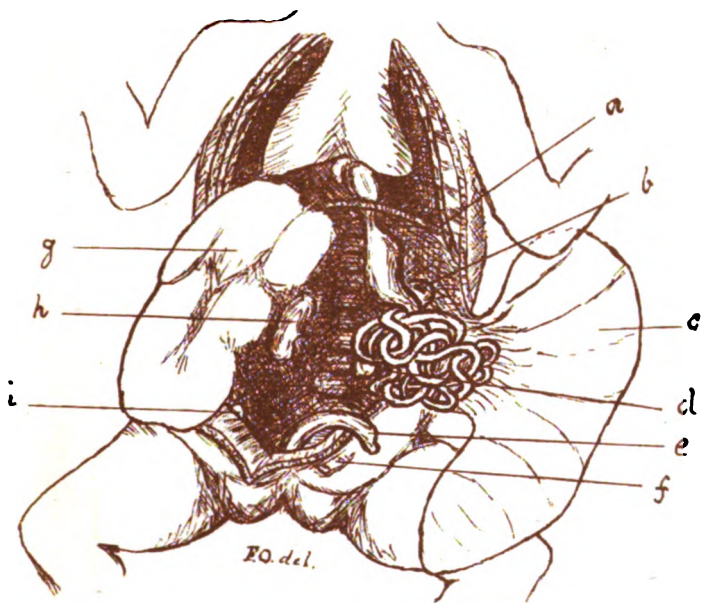




External View  
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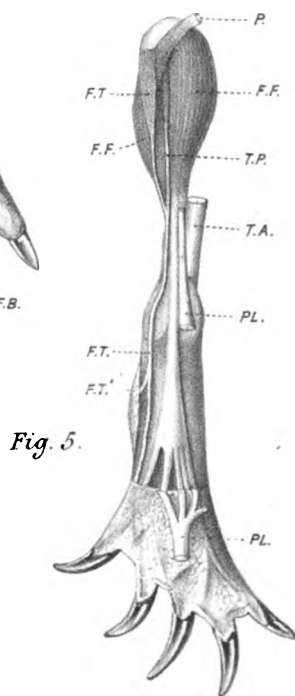
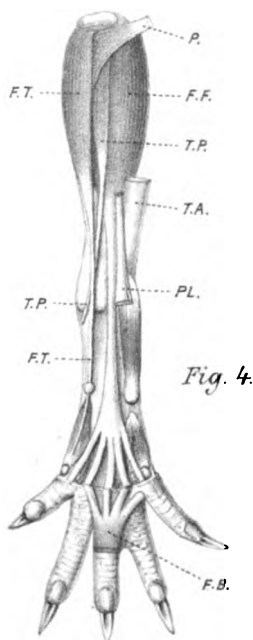
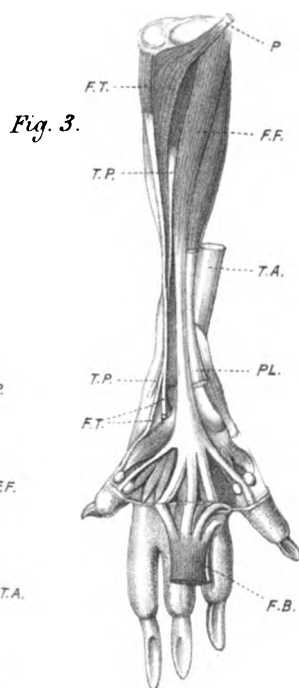
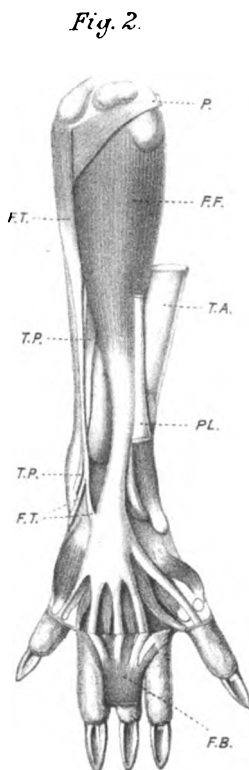
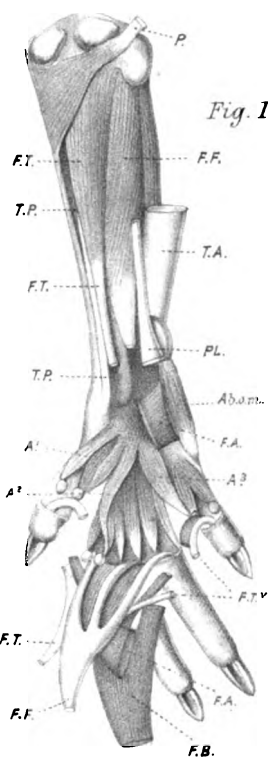




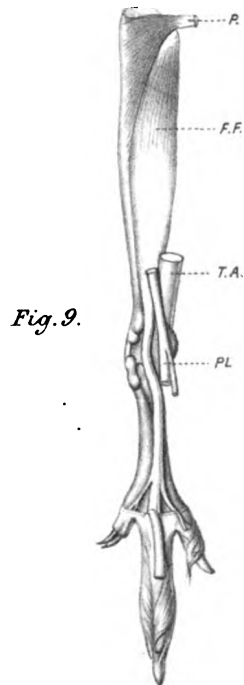
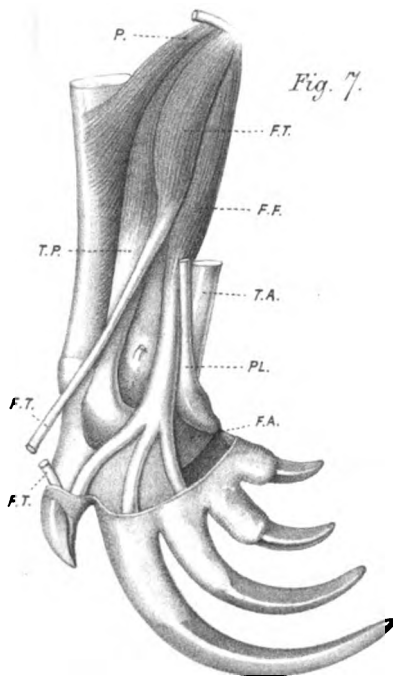
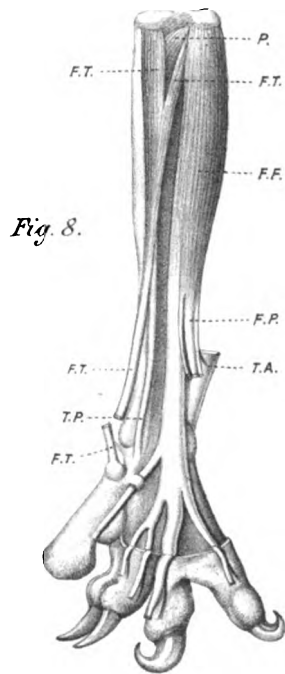
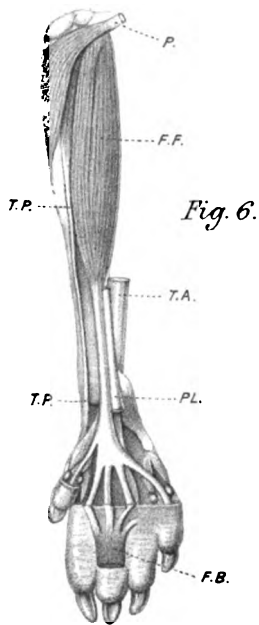


Internal View  
II











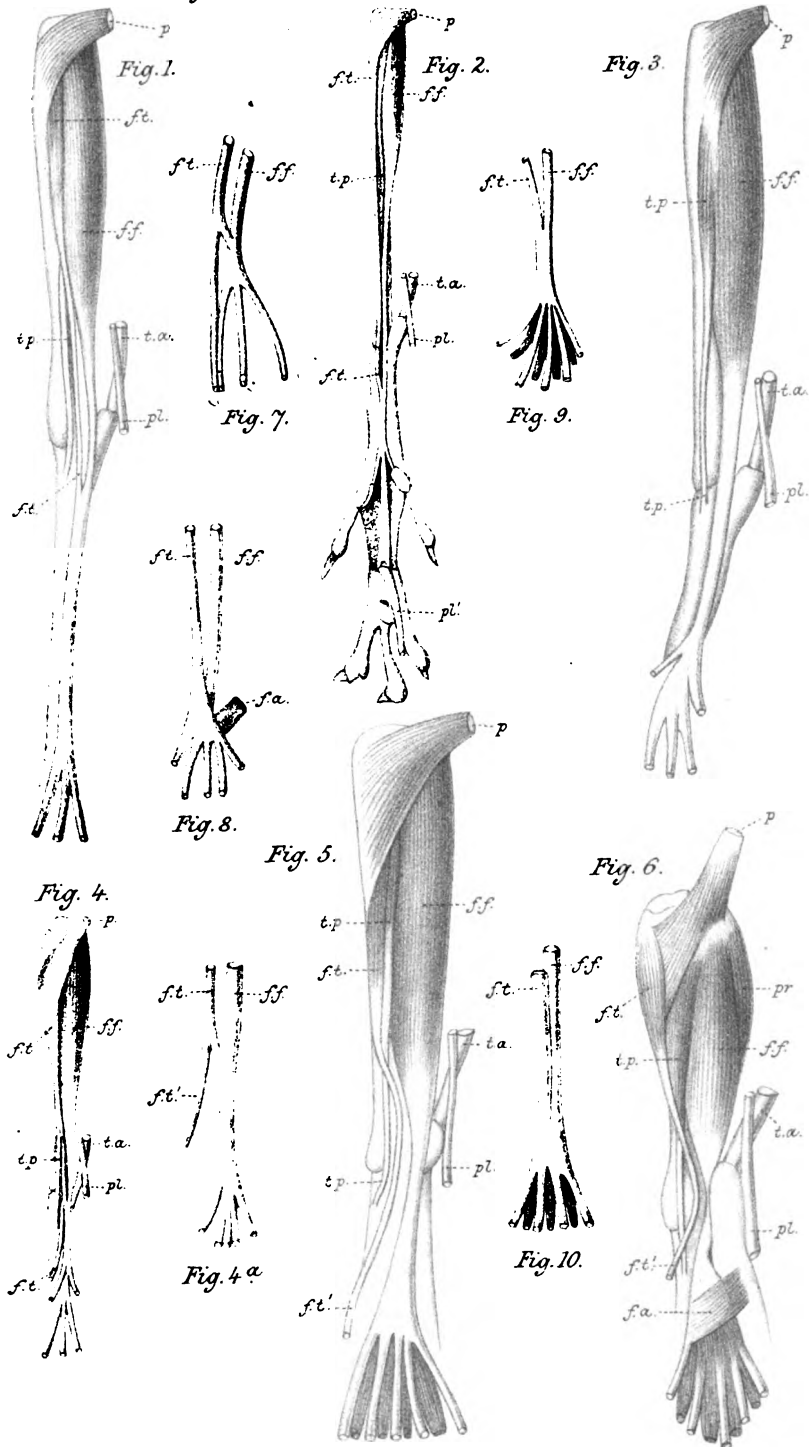


PLATE VI.

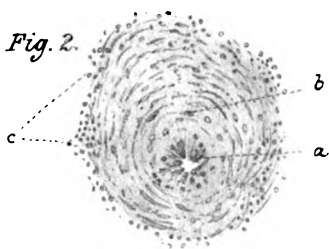


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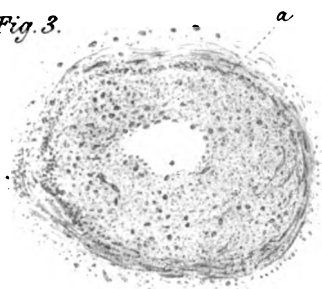
*Fig. 1.*



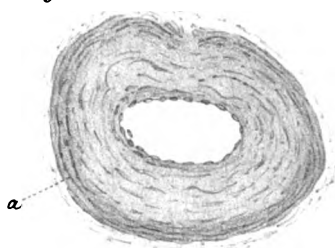
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*Fig. 3.*



*Fig. 4.*



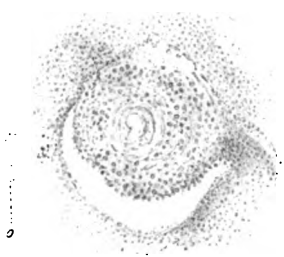
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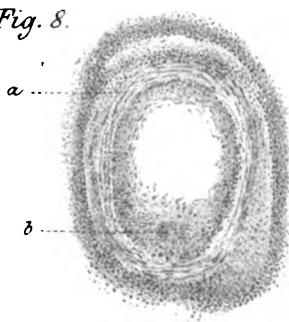
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*Fig. 7.*



*Fig. 8.*

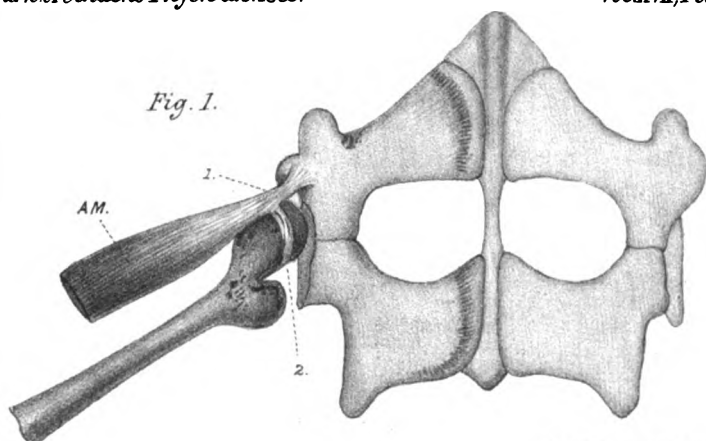


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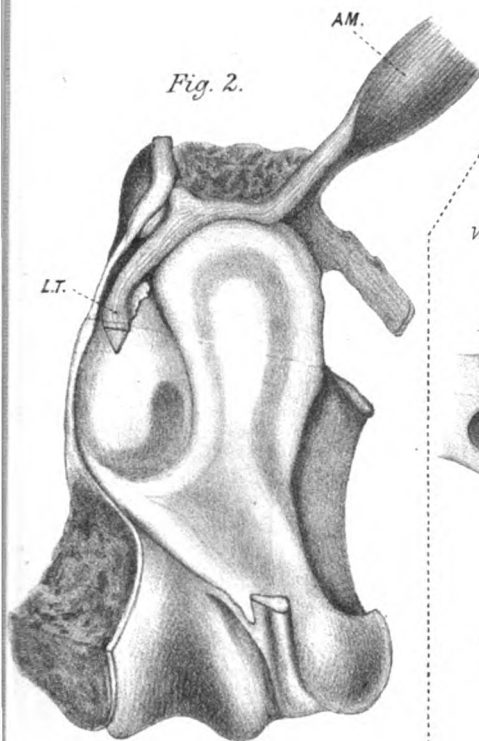
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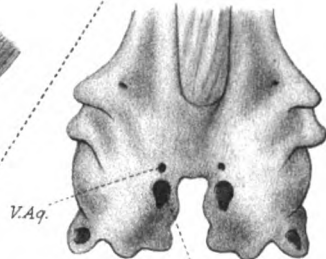
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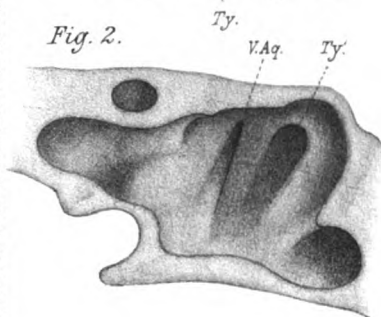
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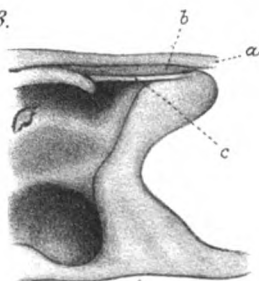
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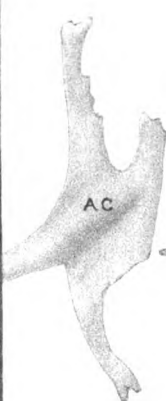


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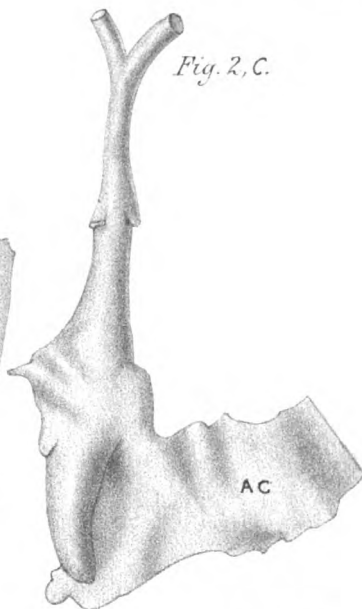




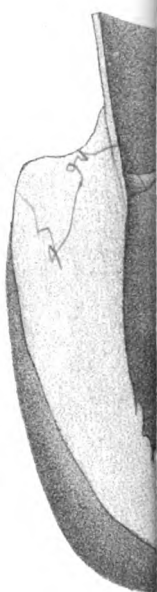
*Fig. 1, A, 1, C.*



*Fig. 2, C.*



*Fig. 4, C.*

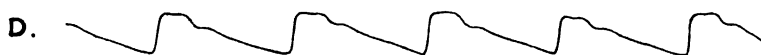


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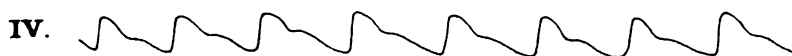
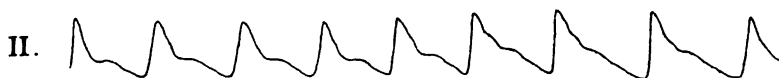




*Exper. CXII.*



*Exper. CXIV.*







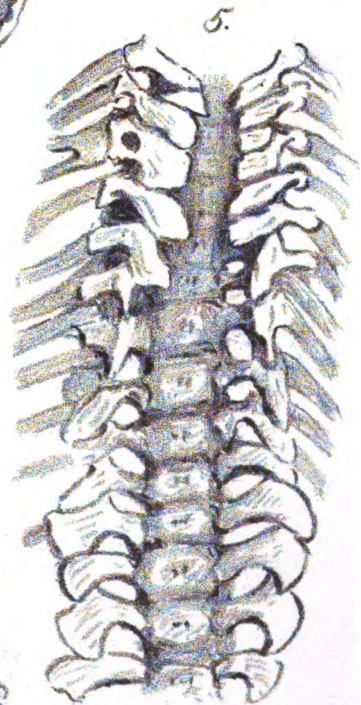
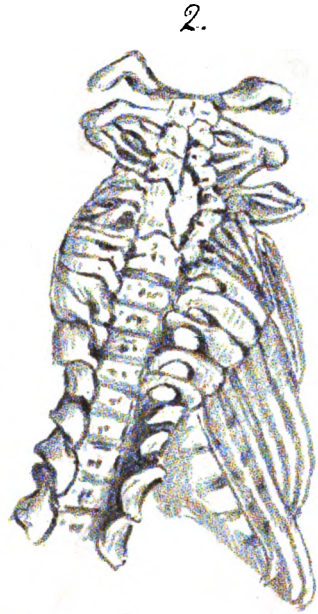














Fig. 1.

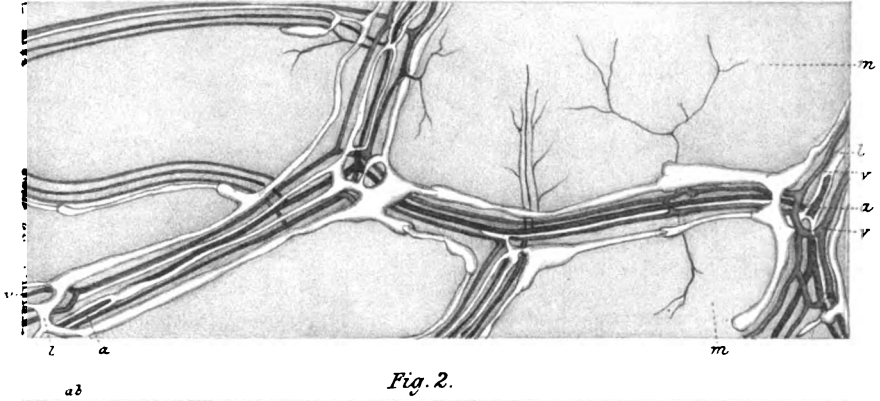


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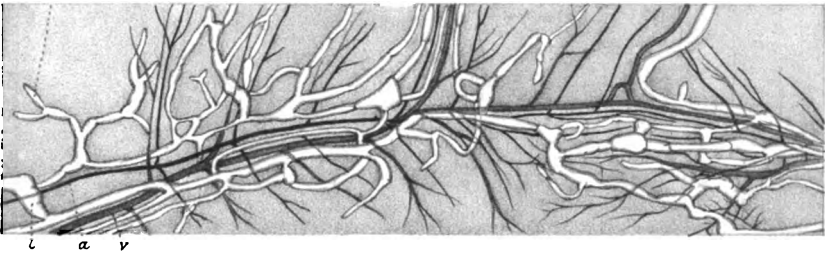


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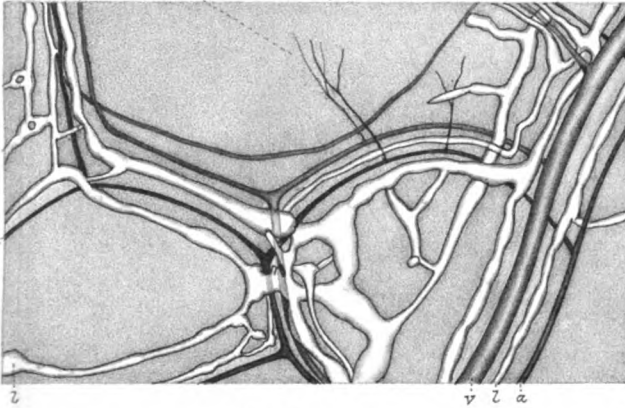


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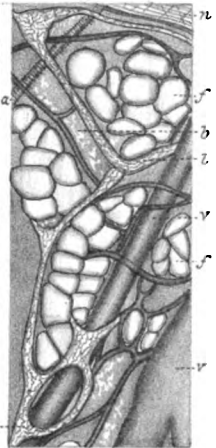


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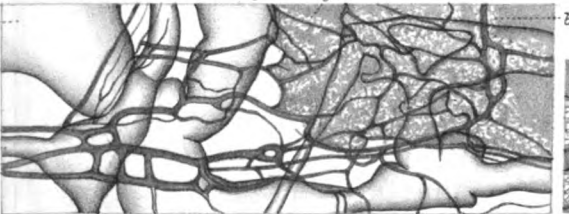
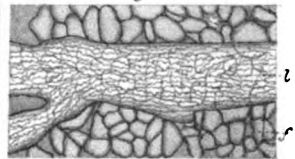


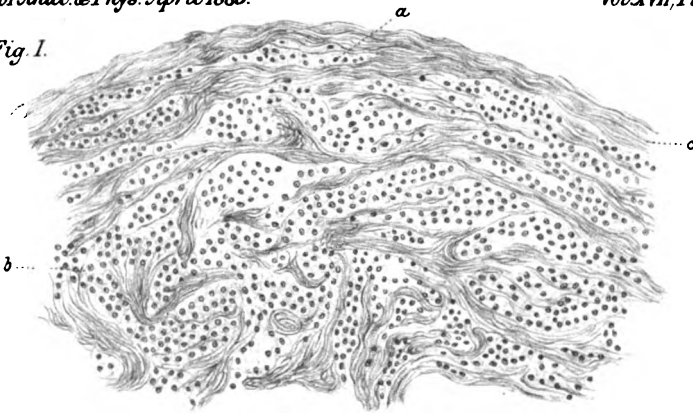
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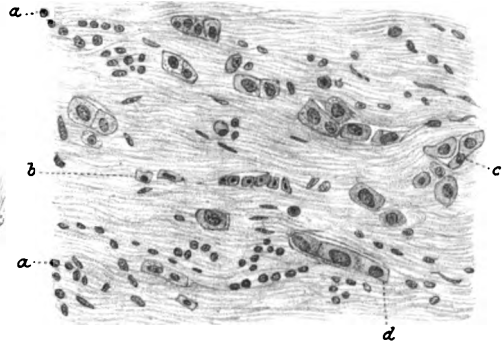
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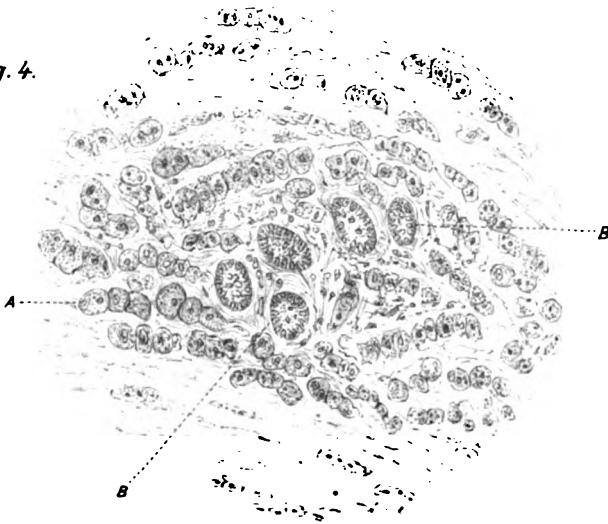
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*Fig. 3.*



*Fig. 4.*

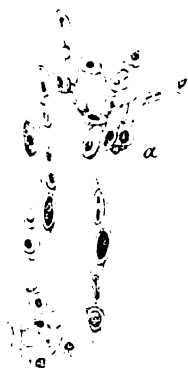




*Fig. 1.*



*Fig. 2.*



*Fig. 3.*



*Fig. 4.*



*Fig. 6.*



*Fig. 8.*



*Fig. 7.*



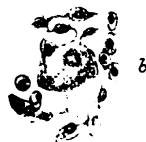
*Fig. 5.*



*Fig. 9.*

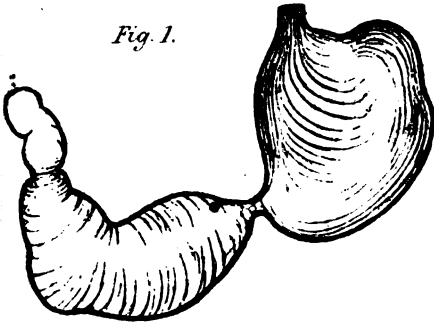


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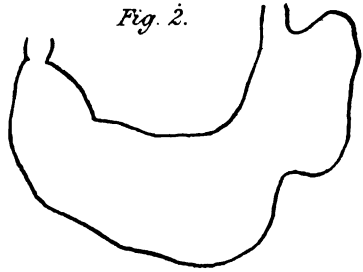




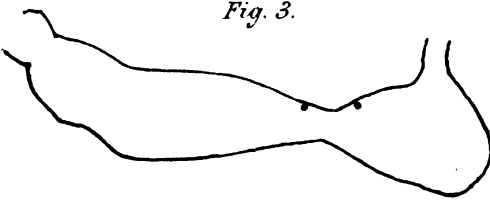
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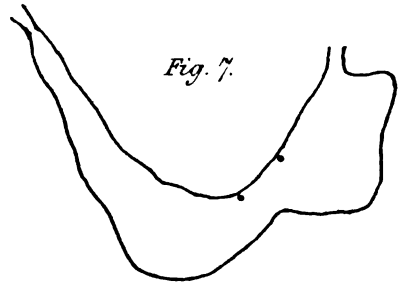
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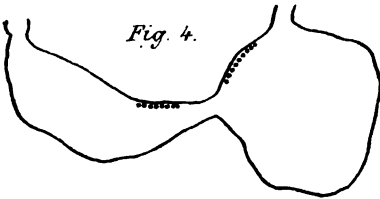
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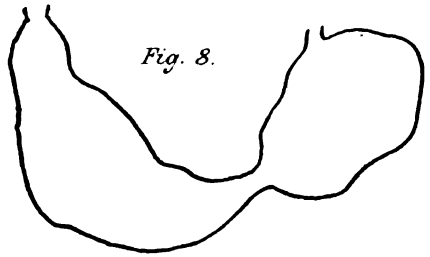
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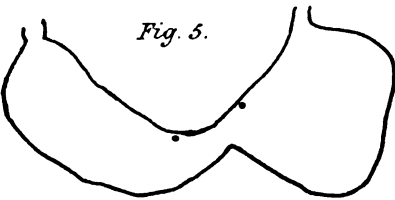
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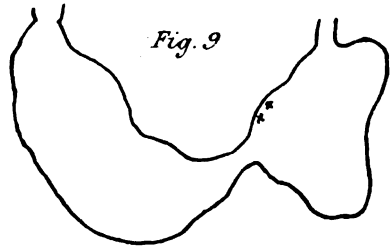
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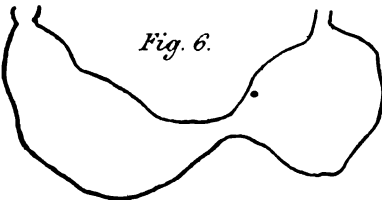
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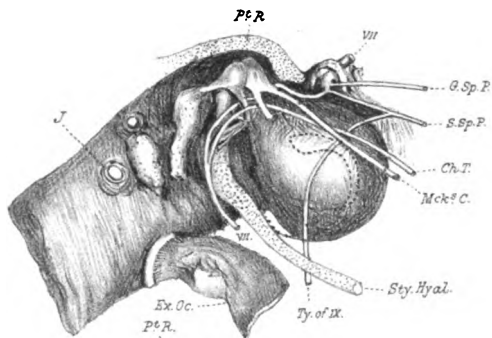


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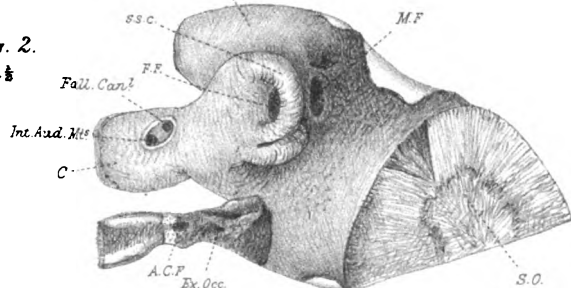


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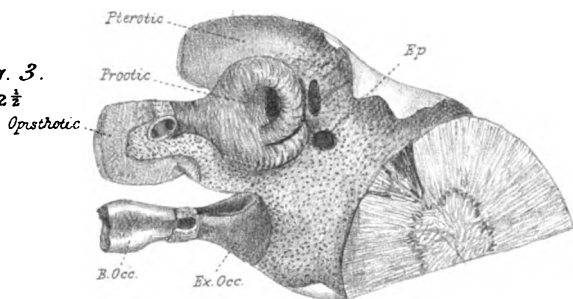
*Fig. 2.*

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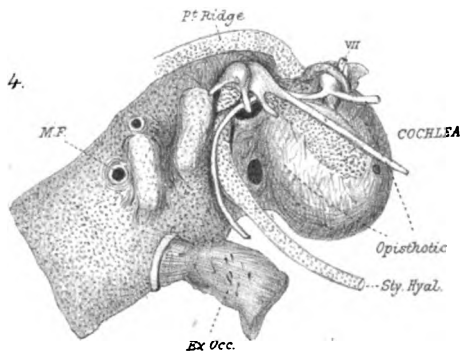


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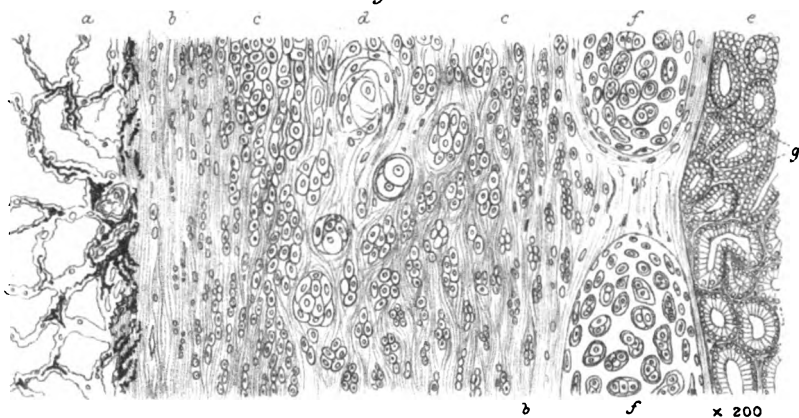
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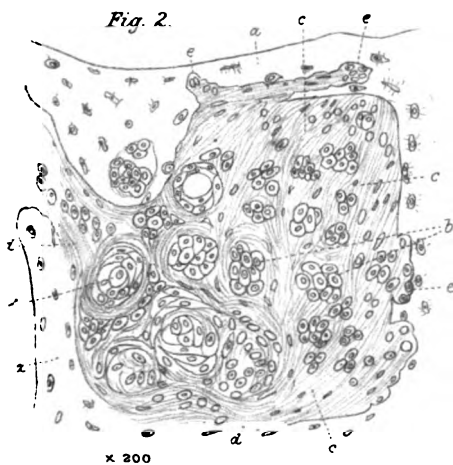




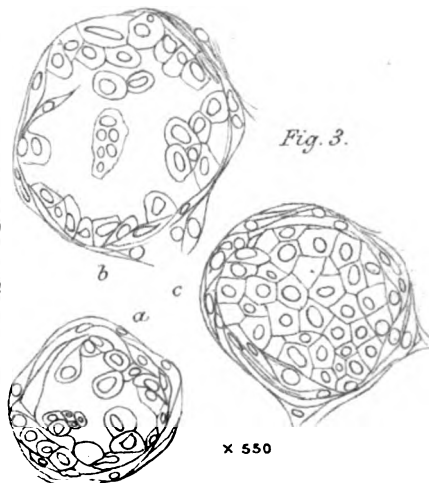
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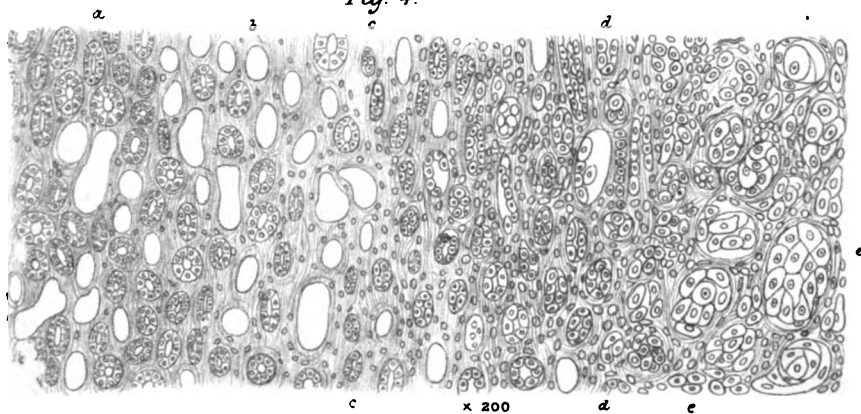
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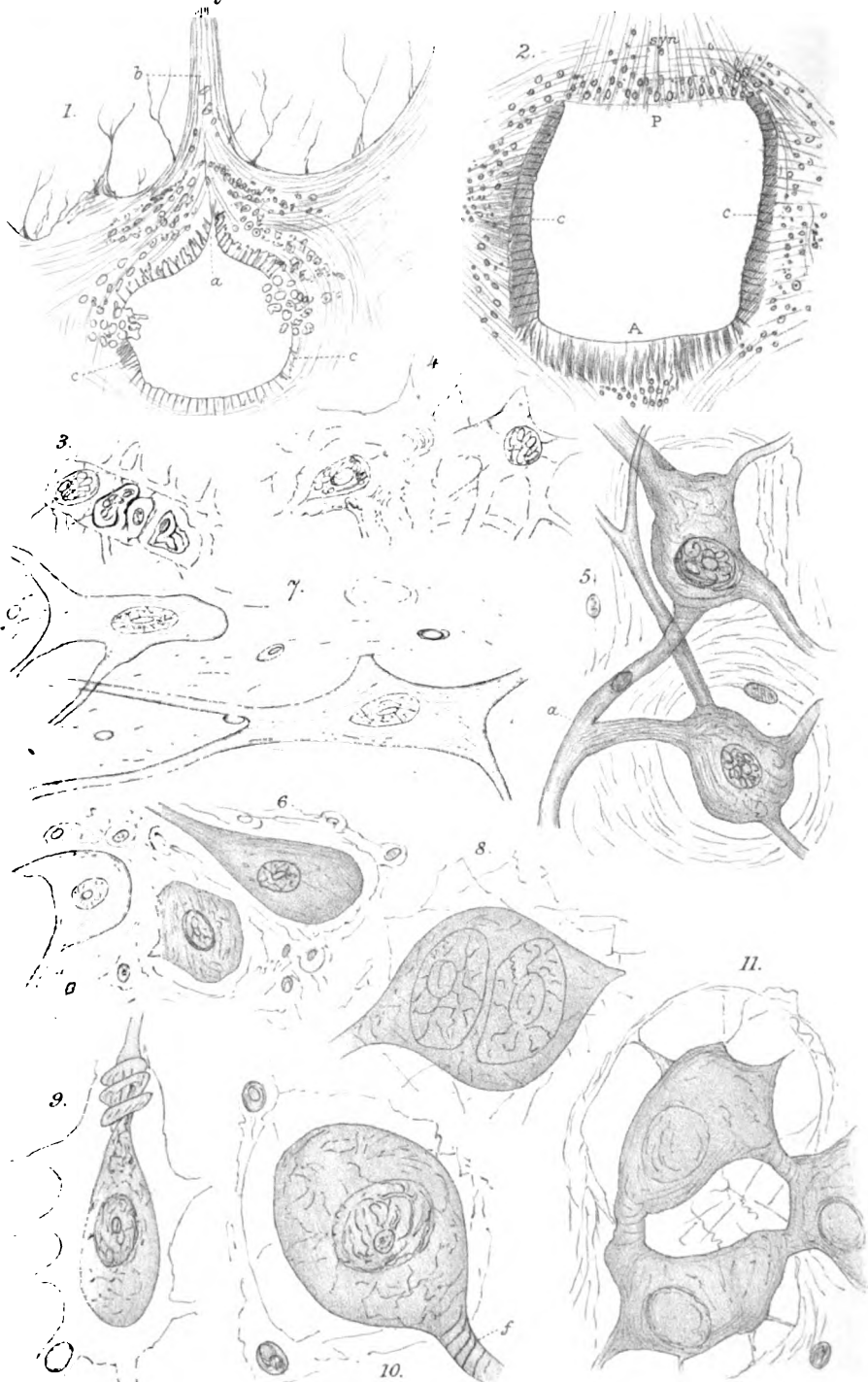
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*Fig. 4.*









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